Baskar, P.
10/649156

FILE 'CAPLUS' ENTERED AT 13:53:59 ON 14 MAR 2006
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FILE COVERS 1907 - 14 Mar 2006 VOL 144 ISS 12
FILE LAST UPDATED: 13 Mar 2006 (20060313/ED)

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L1 0 S PTA2204 OR PTA 2204

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FILE 'CONFSCI' ENTERED AT 13:53:59 ON 14 MAR 2006

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FILE 'JICST-EPLUS' ENTERED AT 13:53:59 ON 14 MAR 2006

FILE 'JAPIO' ENTERED AT 13:53:59 ON 14 MAR 2006

L2 0 S L1

FILE 'USPATFULL' ENTERED AT 13:54:00 ON 14 MAR 2006

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 14 Mar 2006 (20060314/PD)
FILE LAST UPDATED: 14 Mar 2006 (20060314/ED)
HIGHEST GRANTED PATENT NUMBER: US7013485
HIGHEST APPLICATION PUBLICATION NUMBER: US2006053519
CA INDEXING IS CURRENT THROUGH 14 Mar 2006 (20060314/UPCA)

Searcher : Shears 571-272-2528

ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 14 Mar 2006 (20060314/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Dec 2005
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Dec 2005

L3 2 S L1

L3 ANSWER 1 OF 2 USPATFULL on STN
ACCESSION NUMBER: 2004:50916 USPATFULL
TITLE: Novel human protein kinases and uses therefor
INVENTOR(S): Meyers, Rachel, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004038346	A1	20040226
APPLICATION INFO.:	US 2003-649156	A1	20030827 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2001-799875, filed on 6 Mar 2001, GRANTED, Pat. No. US 6638721
Continuation-in-part of Ser. No. US 2000-659289, filed on 12 Sep 2000, GRANTED, Pat. No. US 6518216

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-182059P	20000211 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 58 Drawing Page(s)
LINE COUNT: 6019
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to novel kinase nucleic acid sequences and proteins. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 2 OF 2 USPATFULL on STN
ACCESSION NUMBER: 2002:60946 USPATFULL
TITLE: Novel human protein kinases and uses therefor
INVENTOR(S): Meyers, Rachel, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002034780	A1	20020321
	US 6638721	B2	20031028
APPLICATION INFO.:	US 2001-799875	A1	20010306 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-659287, filed on 12 Sep 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-182059P	20000211 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000	
NUMBER OF CLAIMS:	22	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	58 Drawing Page(s)	
LINE COUNT:	6018	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to novel kinase nucleic acid sequences and proteins. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 13 MAR 2006 HIGHEST RN 876655-59-3
DICTIONARY FILE UPDATES: 13 MAR 2006 HIGHEST RN 876655-59-3

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TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

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* *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added, *
* effective March 20, 2005. A new display format, IDERL, is now *
* available and contains the CA role and document type information. *
* *
****

Structure search iteration limits have been increased. See HELP SLIMITS for details.

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http://www.cas.org/ONLINE/UG/regprops.html

		E	SODIUM CHLORIDE/CN
L4	445	S	SODIUM CHLORIDE ?/CN
		E	SODIUM CITRATE/CN
L5	5	S	E3-7

Searcher : Shears 571-272-2528

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L6          450 S L4 OR L5
                E PROTEIN KINASE/CN
L7         1698 S PROTEIN KINASE ?/CN

     FILE 'CAPLUS' ENTERED AT 13:55:14 ON 14 MAR 2006
L4          445 SEA FILE=REGISTRY ABB=ON  PLU=ON  SODIUM CHLORIDE ?/CN
L5            5 SEA FILE=REGISTRY ABB=ON  PLU=ON  ("SODIUM CITRATE"/CN OR
                  "SODIUM CITRATE (NA2O7C6H6)"/CN OR "SODIUM CITRATE
                  (NA3C6D5O7)"/CN OR "SODIUM CITRATE (NA3C6H5O7)"/CN OR
                  "SODIUM CITRATE (NAC6H7O7)"/CN)
L7         1698 SEA FILE=REGISTRY ABB=ON  PLU=ON  PROTEIN KINASE ?/CN
L8       165813 SEA FILE=CAPLUS ABB=ON  PLU=ON  L7 OR PROTEIN KINASE
L9       430653 SEA FILE=CAPLUS ABB=ON  PLU=ON  L4 OR (NA OR SODIUM)(W)(CL
                  OR CHLORIDE) OR NACL OR SALINE
L10       20942 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 OR (NA OR TRISODIUM OR
                  SODIUM)(W)CITRATE OR CITRA
L11        1634 SEA FILE=CAPLUS ABB=ON  PLU=ON  L8 AND L9
L12           5 SEA FILE=CAPLUS ABB=ON  PLU=ON  L11 AND L10



L4          445 SEA FILE=REGISTRY ABB=ON  PLU=ON  SODIUM CHLORIDE ?/CN
L5            5 SEA FILE=REGISTRY ABB=ON  PLU=ON  ("SODIUM CITRATE"/CN OR
                  "SODIUM CITRATE (NA2O7C6H6)"/CN OR "SODIUM CITRATE
                  (NA3C6D5O7)"/CN OR "SODIUM CITRATE (NA3C6H5O7)"/CN OR
                  "SODIUM CITRATE (NAC6H7O7)"/CN)
L7         1698 SEA FILE=REGISTRY ABB=ON  PLU=ON  PROTEIN KINASE ?/CN
L8       165813 SEA FILE=CAPLUS ABB=ON  PLU=ON  L7 OR PROTEIN KINASE
L9       430653 SEA FILE=CAPLUS ABB=ON  PLU=ON  L4 OR (NA OR SODIUM)(W)(CL
                  OR CHLORIDE) OR NACL OR SALINE
L10       20942 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 OR (NA OR TRISODIUM OR
                  SODIUM)(W)CITRATE OR CITRA
L13        1642 SEA FILE=CAPLUS ABB=ON  PLU=ON  L8 AND (L9 OR L10)
L14          29 SEA FILE=CAPLUS ABB=ON  PLU=ON  L13 AND (HYBRIDIS? OR
                  HYBRIDIZ?)

L16          32 L12 OR L14

L16  ANSWER 1 OF 32  CAPLUS  COPYRIGHT 2006 ACS on STN
ED   Entered STN:  08 Jun 2005
ACCESSION NUMBER:         2005:482477  CAPLUS
DOCUMENT NUMBER:          143:53914
TITLE:                    Hepatic leptin signaling in obesity
AUTHOR(S):                Brabant, Georg; Mueller, Guenter; Horn, Ruediger;
                          Roden, Michael; Nave, Heike
CORPORATE SOURCE:         Department of Gastroenterology, Hepatology and
                          Endocrinology, Hannover Medical School, Hannover,
                          30625, Germany
SOURCE:                   FASEB Journal (2005), 19(8), 1048-1050,
                          10.1096/fj.04-2846fje
                          CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER:                Federation of American Societies for Experimental
                          Biology
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Obesity, a state of apparent "leptin resistance," is well known to be
     associated with insulin resistance.  In diet-induced obesity (DIO),
     hepatic insulin signaling is impaired but the link between leptin and
     insulin signaling pathways is only incompletely defined.  This study
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was to evaluate the effects of DIO on leptin and insulin cross-signaling in the liver. Leptin receptor expression was measured by in situ **hybridization** with pan-leptin receptor probes and by immunoblotting. Furthermore, intracellular signaling was investigated in vivo under basal conditions and at 45 and 360 min after stimulation with a bolus of human recombinant leptin (hrec-leptin; 1 mg/kg body wt) or **saline**. At baseline, all forms of the leptin receptor were markedly to completely down-regulated in DIO rats. Hrec-leptin bolus injection stimulated leptin-dependent signaling with a fivefold increase in JAK-2pY in lean but not in DIO rats. Basal IRpY, IRS-1pY, IRS-1p85, IRS-2pY, IRSp85, and PKBpT308 levels were reduced ($P<0.01$) in DIO rats as compared with lean controls. Basal GSK-3β serine phosphorylation (S9) was higher ($P < 0.01$) in lean animals along with lower basal PEPCK activity compared with DIO rats consistent with the insulin and leptin resistance of the latter. Only in lean animals phosphorylation of PKB (T308) and GSK-3β (S9) was acutely stimulated by leptin at 45 min followed by inhibition at 6 h after application. AMPKα protein levels as well as basal and leptin-stimulated total and α-specific AMPK activity were comparable in both groups. These data show that in a model of dietary-induced obesity (1) leptin receptors and subsequent signaling events are down-regulated, (2) basal insulin signaling is impaired, and (3) the cross-talk between leptin and insulin signaling is differentially regulated by the nutritional status, which is sensed by AMPK in rat liver. Thus, the liver seems to play a major role in the modulation of the leptin signal and insulin resistance in obesity.

IT **148640-14-6, Protein kinase** B
152478-57-4, JAK2 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(effects of diet-induced obesity on leptin and insulin cross-signaling in the liver)

IT **443900-95-6,** GSK-3β
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hepatic leptin and insulin cross signaling in diet-induced obesity)

IT **142008-29-5, Protein kinase** A
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α; effects of diet-induced obesity on leptin and insulin cross-signaling in the liver)

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 2 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 03 May 2005
ACCESSION NUMBER: 2005:377842 CAPLUS
DOCUMENT NUMBER: 143:453706
TITLE: Analysis of salt-stress-inducible ESTs isolated by PCR-subtraction in salt-tolerant rice
AUTHOR(S): Shiozaki, Noriko; Yamada, Mika; Yoshiba, Yoshu
CORPORATE SOURCE: Life Science Research Center, Central Research Laboratory, Hitachi Ltd., Hatoyama, 350-0395, Japan
SOURCE: Theoretical and Applied Genetics (2005), 110(7), 1177-1186
CODEN: THAGA6; ISSN: 0040-5752
PUBLISHER: Springer GmbH
DOCUMENT TYPE: Journal

LANGUAGE: English

AB To clarify the mechanisms of stress tolerance in rice and to search for rice genes associated with these mechanisms, we analyzed genes induced by a high salinity treatment using the PCR-subtractive **hybridization** method (PCR-subtraction). Seedlings of the salt-tolerant rice cultivar Dee-geo-woo-gen (DGWG) were either treated with 250 mM **NaCl** for 5 h or left untreated, and PCR-subtraction was then performed using the untreated (control) plants as a driver and the **NaCl**-treated plants as a tester. We obtained 384 clones of tester-specific cDNAs as salt-inducible candidates. Northern anal. performed with the cDNA fragments showed that 65 clones had been induced by the **NaCl** treatment. Sequence anal. and database searching indicated that these clones have homol. to proteins functional for detoxification, stress response, and signal transduction in plants. Of these clones, 22% coded for unknown proteins and 12% gave no hits. We selected eight clones from each functional category and analyzed their expression pattern in DGWG. For temporal anal., seedlings were treated with H2O or 250 mM **NaCl** for 0, 0.5, 1, 2, 5, 10 or 24 h. Different patterns of transcript regulation were found. For the anal. of expression in response to various types of stress and abscisic acid (ABA) treatments, seedlings were treated for 5 h or 10 h with H2O, dehydration, cold (4°C), heat (40°C), mannitol, ABA, or wounding. All clones were strongly up-regulated by osmotic stress (dehydration and mannitol) and the ABA treatment.

IT **7647-14-5, Sodium chloride**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(anal. of salt-stress-inducible ESTs isolated by PCR-subtraction in salt-tolerant rice)

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 3 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 02 May 2005
ACCESSION NUMBER: 2005:373501 CAPLUS
DOCUMENT NUMBER: 143:146393
TITLE: Regulation of G protein-coupled receptor kinase 5 mRNA and protein level in rat brain by addictive drugs
AUTHOR(S): Zhu, Min; Fan, Xueliang; Yang, Weilin; Jiang, Yan; Ma, Lan
CORPORATE SOURCE: Shanghai Medical College, Fudan University, Shanghai, 200032, Peop. Rep. China
SOURCE: Shengli Xuebao (2004), 56(5), 559-565
CODEN: SLHPAH; ISSN: 0371-0874
PUBLISHER: Kexue Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The current study investigated the potential effects of acute administration of addictive drugs including morphine, heroine and cocaine on G protein-coupled receptor kinase 5 (GRK5) mRNA level in the rat brain using in situ **hybridization** and analyzed the effects of acute and chronic morphine treatments on GRK5 protein level in the rat brain using Western blotting assay. Our results showed that 2 h after the initial morphine (10 mg/kg), cocaine (15 mg/kg) and heroine (1 mg/kg) treatment, the mRNA level of GRK5 in the parietal cortex increased about 110% (P<0.01), 70% (P<0.05) and 100% (P<0.01),

resp. In the temporal cortex, GRK5 mRNA level increased about 90% (P<0.01), 40% (P<0.05) and 80.0% (P<0.01), resp. In the hippocampus, the mRNA level of GRK5 increased about 60% (P<0.01), 30% (P<0.05) and 80% (P<0.01). However, the mRNA level of GRK5 remained unchanged after acute morphine, cocaine or heroine treatment. In the cerebral cortex of rat brain, the acute administration of morphine (NS-Mor) increased GRK5 protein level by about 60% while the chronic morphine treatment (Mor-Mor) increased GRK5 protein level even higher [about 130% compared with the control group (chronic **saline** treatment, NS-NS) group, P<0.01]. In the hippocampus, GRK5 protein level remained unchanged after acute administration of morphine (P>0.1), while the level of GRK5 protein tended to decrease after chronic morphine treatment (P=0.098). In the thalamus, acute morphine treatment caused no change in GRK5 protein level (P>0.1) while after chronic morphine treatment, GRK5 protein level decreased significantly (more than 90%, P<0.01). Taken together, our results indicate that addictive drugs can regulate GRK5 in the rat brain on protein level as well as on mRNA level and suggest that GRK5 may play a role in addiction of psychoactive substances.

IT **153700-57-3**, G Protein-coupled receptor kinase 5
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(regulation of G protein-coupled receptor kinase 5 mRNA and protein level in rat brain by addictive drugs)

L16 ANSWER 4 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 25 Mar 2005
ACCESSION NUMBER: 2005:259767 CAPLUS
DOCUMENT NUMBER: 142:330814
TITLE: Protein and cDNA sequences of casein kinase stress-related proteins (CKSRP) and methods of use for increasing stress tolerance in transgenic plants
INVENTOR(S): Shirley, Amber; Allen, Damian; Van Thielen, Nocha E.; da Costa e Silva, Oswaldo; Chen, Ruoying; Mills, Lori
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 80 pp., Cont.-in-part of U.S. Ser. No. 292,408.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005066396	A1	20050324	US 2004-904588	20041117
US 2002059662	A1	20020516	US 2001-828313	20010406
US 6867351	B2	20050315		
CA 2405697	AA	20020613	CA 2001-2405697	20010406
AU 2002043190	A5	20020618	AU 2002-43190	20010406
EP 1335986	A2	20030820	EP 2001-989068	20010406
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
US 2003182692	A1	20030925	US 2002-292408	20021112
US 2004194163	A1	20040930	US 2003-688481	20031017
US 2004107463	A1	20040603	US 2003-716089	20031118
US 2004148658	A1	20040729	US 2004-764259	20040123
US 2004128721	A1	20040701	US 2004-768511	20040130

US 2004199946	A1	20041007	US 2004-768863		20040130
US 2004216183	A1	20041028	US 2004-770225		20040202
PRIORITY APPLN. INFO.:			US 2000-196001P	P	20000407
			US 2001-828313	A2	20010406
			US 2001-346096P	P	20011109
			US 2002-292408	A2	20021112
			US 2001-828062	A3	20010406
			US 2001-828302	A3	20010406
			US 2001-828303	A3	20010406
			US 2001-828310	A3	20010406
			US 2001-828447	A3	20010406
			WO 2001-US11253	W	20010406

AB The present invention describes a novel type of Casein Kinase Stress-Related Polypeptides (CKSRPs) and CKSRP coding nucleic acids that are important for modulating a plant's response to an environmental stress. More particularly, overexpression of these CKSRP coding nucleic acids in a transgenic plant results in the plant's increased tolerance to an environmental stress. Preferably, the CKSRP is from Physcomitrella patens, Saccharomyces cerevisiae, or Brassica napus. Namely, described herein are the Physcomitrella patens Casein Kinase (CK)-1, CK-2, CK-4, Saccharomyces cerevisiae CK-1, Brassica napus CK-1, CK-2, CK-3, CK-4 and CK-5. Also provided are agricultural products, including seeds, produced by the transgenic plants. Also provided are isolated CKSRPs, and isolated nucleic acid coding CKSRPs, and vectors and host cells containing the latter.

IT **68-04-2, Sodium citrate 7647-14-5, Sodium chloride,** uses
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(6 x SSC solution; protein and cDNA sequences of casein kinase stress-related proteins (CKSRP) and methods of use for increasing stress tolerance in transgenic plants)

IT **52660-18-1,** Casein Kinase 1 **366806-33-9,** Casein Kinase 2
RL: AGR (Agricultural use); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(sequence homolog; protein and cDNA sequences of casein kinase stress-related proteins (CKSRP) and methods of use for increasing stress tolerance in transgenic plants)

L16 ANSWER 5 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 20 Jan 2005
ACCESSION NUMBER: 2005:49963 CAPLUS
DOCUMENT NUMBER: 142:291158
TITLE: Changes in expression of the mouse homologues of KIAA genes after subchronic methamphetamine treatment
AUTHOR(S): Yamamoto, H.; Imai, K.; Takamatsu, Y.; Kamegaya, E.; Hara, Y.; Shimada, K.; Yamamoto, T.; Shen, H.-W.; Hagino, Y.; Kobayashi, H.; Ide, S.; Sora,

I.; Koga, H.; Ikeda, K.
CORPORATE SOURCE: Tokyo Institute of Psychiatry, Tokyo, 156-8585, Japan
SOURCE: Annals of the New York Academy of Sciences (2004), 1025(Current Status of Drug Dependence/Abuse Studies), 92-101
CODEN: ANYAA9; ISSN: 0077-8923
PUBLISHER: New York Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Amphetamine abuse may be associated with adaptive changes in gene expression in the brain. In the present study, a newly developed cDNA array system comprising mouse KIAA (mKIAA) cDNA clones was used to examine the gene expression affected by chronic methamphetamine treatment. Approx. 800 mKIAA clones were blotted onto a nylon membrane and **hybridized** with 33P-labeled cDNA derived from mRNAs isolated from the whole brains of mice that had been treated daily with **saline** or methamphetamine (2 mg/kg, i.p.) for 2 wk. The arrays displayed robust **hybridization** for almost all transcripts. The results obtained from five expts. were averaged, each performed with triplicate samples. Several clones were chosen as pos. candidates for methamphetamine-induced changes; however, only Per2 and mKIAA0099 genes showed a significantly increased expression ($P < .05$). Subsequently, with the focus on the period-related proteins, the expression of these proteins in various parts of the rat brain were assessed by immunoblot anal. Chronic administration of methamphetamine (8 mg/kg, i.p., for 10 days) caused increased Per2 protein expression in the hippocampus. Interestingly, chronic methamphetamine treatment at a lower dose (4 mg/kg, i.p., for 10 days) induced an increase in SCN circadian oscillatory protein (SCOP) expression, also in the hippocampus. These data suggest that long-lasting alterations of the period-related gene expressions in the hippocampus might play an important role in methamphetamine addiction.

IT **137632-07-6**, Extracellular signal-regulated **protein kinase** 1
RL: BSU (Biological study, unclassified); BIOL (Biological study) (chronic treatment with methamphetamine had no significant effect on extracellular signal-regulated **protein kinase** 1 immunoreactivity level in any brain area in mouse)

IT **137632-08-7**, Extracellular signal-regulated **protein kinase** 2
RL: BSU (Biological study, unclassified); BIOL (Biological study) (chronic treatment with methamphetamine had no significant effect on extracellular signal-regulated **protein kinase** 2 immunoreactivity level in any brain area in mouse)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 6 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 13 Sep 2004
ACCESSION NUMBER: 2004:746343 CAPLUS
DOCUMENT NUMBER: 141:254382
TITLE: L-type Ca2+ channels mediate adaptation of extracellular signal-regulated kinase 1/2 phosphorylation in the ventral tegmental area after chronic amphetamine treatment
AUTHOR(S): Rajadhyaksha, Anjali; Husson, Isabelle; Satpute, Shirish S.; Kueppenbender, Karsten D.; Ren, J. Q.;

Guerriero, Rejean M.; Standaert, David G.; Kosofsky, Barry E.
CORPORATE SOURCE: NMR Center, Department of Radiology, Harvard Medical School, Charlestown, MA, 02129, USA
SOURCE: Journal of Neuroscience (2004), 24(34), 7464-7476
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB L-type Ca2+ channels (LTCCs) play an important role in chronic psychostimulant-induced behaviors. However, the Ca2+ second messenger pathways activated by LTCCs after acute and recurrent psychostimulant administration that contribute to drug-induced mol. adaptations are poorly understood. Using a chronic amphetamine treatment paradigm in rats, the authors have examined the role of LTCCs in activating the mitogen-activated protein (MAP) kinase pathway in the ventral tegmental area (VTA), a primary target for the reinforcing properties of psychostimulants. Using immunoblot and immunohistochem. analyses, the authors find that in chronic **saline**-treated rats a challenge injection of amphetamine increases phosphorylation of MAP [extracellular signal-regulated kinase 1/2 (ERK1/2)] kinase in the VTA that is independent of LTCCs. However, in chronic amphetamine-treated rats there is no increase in amphetamine-mediated ERK1/2 phosphorylation unless LTCCs are blocked, in which case there is robust phosphorylation in VTA dopamine neurons. Examination of the expression of phosphatases reveals an increase in calcineurin [protein phosphatase 2B (PP2B)] and MAP kinase phosphatase-1 (MKP-1) in the VTA. Using in situ **hybridization** histochem. and immunoblot analyses, the authors further examined the mRNA and protein expression of the LTCC subtypes Cav1.2 and Cav1.3 in VTA dopamine neurons in drug-naive animals and in rats after chronic amphetamine treatment. The authors found an increase in Cav1.2 mRNA and protein levels, with no change in Cav1.3. Together, our results suggest that one aspect of LTCC-induced changes in second messenger pathways after chronic amphetamine exposure involves activation of the MAP kinase phosphatase pathway by upregulation of Cav1.2 in VTA dopaminergic neurons.

IT **137632-07-6, Protein kinase** ERK1
137632-08-7, Protein kinase ERK2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(L-type Ca2+ channels mediate adaptation of extracellular signal-regulated kinase 1/2 phosphorylation in ventral tegmental area after chronic amphetamine treatment)

REFERENCE COUNT: 94 THERE ARE 94 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 7 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 22 Oct 2003
ACCESSION NUMBER: 2003:828838 CAPLUS
DOCUMENT NUMBER: 140:192853
TITLE: Elevated neuronal nitric oxide synthase expression in chronic haloperidol-treated rats
AUTHOR(S): Lau, Yuen-Sum; Petroske, Elizabeth; Meredith, Gloria E.; Wang, John Q.
CORPORATE SOURCE: School of Pharmacy, Division of Pharmacology, University of Missouri-Kansas City, Kansas City, MO, 2411, USA
SOURCE: Neuropharmacology (2003), 45(7), 986-994
CODEN: NEPHBW; ISSN: 0028-3908

PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Long-term administration of neuroleptic drugs, such as haloperidol, in the management of psychiatric disorders may adversely cause an irreversible neurol. syndrome of tardive dyskinesia, which is associated with dopamine (DA2) receptor supersensitivity in the basal ganglia. Recent studies also indirectly suggest an involvement of nitric oxide synthase (NOS) in dopaminergic supersensitivity; however, chronic neuroleptic effects on neuronal NOS (nNOS) expression in the basal ganglia were not reported. In this investigation, the authors treated rats with **saline** or haloperidol (1 mg/kg, s.c.) daily for 21 days. Five days later, the authors detected a significant increase of NOS activity in the striatum of haloperidol-treated rats when compared to **saline** controls. This effect was associated with elevated levels of nNOS mRNA and protein expression in the striatum, but not in the nucleus accumbens, as evidenced by the use of in situ **hybridization**, Western blot, and immunohistochem. techniques. The involvement of the nNOS system after chronic haloperidol treatment coincides with increases of striatal DA2 receptor sites, calmodulin kinase II activity, animal locomotor and stereotypy behaviors. This study suggests an integral role between nNOS, DA2 receptor, and calmodulin system in the development of dopaminergic behavioral supersensitivity resulting from chronic neuroleptic drug treatment. Furthermore, the toxic effect of chronic haloperidol on NOS system selectivity takes place in the neostriatum.
IT **475489-73-7**, Calmodulin kinase II
RL: BSU (Biological study, unclassified); BIOL (Biological study) (elevated neuronal NOS expression in chronic haloperidol-treated rats)
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 8 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 10 Jul 2003
ACCESSION NUMBER: 2003:524101 CAPLUS
DOCUMENT NUMBER: 139:31192
TITLE: Neurokinin 1 receptors and neprilysin modulation of mouse bladder gene regulation
AUTHOR(S): Dozmorov, Igor; Saban, Marcia R.; Gerard, Norma P.; Lu, Bao; Nguyen, Ngoc-Bich; Centola, Michael; Saban, Ricardo
CORPORATE SOURCE: Oklahoma Medical Research Foundation, Microarray Research Facility, Univ. of Oklahoma Health Sciences Center, Oklahoma City, OK, 73104, USA
SOURCE: Physiological Genomics (2003), 12(3), 239-250
CODEN: PHGEFP; ISSN: 1094-8341
URL: http://physiolgenomics.physiology.org/cgi/reprint/12/3/239.pdf
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Neurokinin 1 (NK1) receptors play a fundamental role in neurogenic inflammation. the authors sought to determine the mechanisms downstream from NK1 receptor (NK1R)activation using cDNA arrays and a novel statistical method to analyze gene expression. The authors used female NK1R-/- and wild-type (WT) mice that were sensitized actively by i.p. injections of dinitrophenol 4 (DNP4)-human serum albumin.

Cystitis was induced by intravesical instillation of antigen of DNP4-ovalbumin, and control mice were challenged with **saline** . At 1, 4, and 24 h after instillation,bladders were removed for RNA extraction (n = 3), replicate of RNA extraction (n = 3), and morphol. anal. (n = 6). For cDNA array expts., three bladders from each group were homogenized, and total RNA was obtained. DNase-treated RNA was reverse-transcribed to cDNA, labeled with [α-32P]dATP and **hybridized** to Atlas Mouse 1.2 Arrays. After calculating the mean and SD for background spots, each exptl. value was assigned a normalized score S using the formula S' = (S - Av)/SD, where S' is the original pixel value, and Av and SD are the mean and standard deviation of background spots, resp. Only genes that expressed 3 SD values above background were used. Hypervariable genes were sorted by cluster anal. Matrixes of correlation coeffs. were calculated and represented in a connectivity mosaic. As results, the authors found that in WT mice the most prominent gene cluster had neprilysin in a central position and pos. correlated to a group of activator protein-1 (AP-1)-responsive genes, including laminin-α3, tissue plasminogen activator 11, fos-B, and TNF-β. In WT mice, antigen-induced bladder inflammation led to a downregulation in neprilysin expression. In contrast, NK1R-/- mice failed to mount an inflammatory reaction and presented neprilysin neg. correlated with the same genes described in WT. In conclusion, this work indicates an overriding participation of NK1R and neprilysin in bladder inflammation, provides a working model for the involvement of AP-1 transcription factor, and evokes testable hypotheses regarding the role of NK1R and neprilysin in inflammation.

IT **408328-74-5**, IKKγ kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (neurokinin 1 receptors and neprilysin modulation of mouse bladder gene regulation in relation to gene expression study by cDNA microarray technol.)

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 9 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 13 Sep 2002
ACCESSION NUMBER: 2002:696145 CAPLUS
DOCUMENT NUMBER: 137:227653
TITLE: Genetic engineering of dimorphic fungi for improved secretion of recombinant proteins
INVENTOR(S): Wolff, Anne Mette; Appel, Karen Fuglede; Petersen, Jesper Breum; Poulsen, Ulla; Arnau, Jose; Jacobsen, Mette Dorph
PATENT ASSIGNEE(S): Bioteknologisk Institut, Den.
SOURCE: PCT Int. Appl., 296 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070721	A2	20020912	WO 2002-DK157	20020308
WO 2002070721	A3	20021107		
WO 2002070721	C1	20040429		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003134353	A1	20030717	US 2002-92947		20020308
EP 1379666	A2	20040114	EP 2002-708250		20020308
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
NZ 528597	A	20050225	NZ 2002-528597		20020308
ZA 2003007645	A	20041117	ZA 2003-7645		20030930
PRIORITY APPLN. INFO.:			DK 2001-395	A	20010308
			US 2001-274650P	P	20010312
			WO 2002-DK157	W	20020308

AB It is an object of the present invention to provide fungal host organisms capable of expressing recombinant proteins while at the same time exhibiting satisfactory growth characteristics. It is a further object to provide in a single fungal host organism the characteristic of homogeneous growth and low viscosity typically associated with yeast organisms, and the capability for high protein secretion normally associated with filamentous fungi. It is yet a further object of the invention to provide useful tools for genetic anal. in zygomycetes, including dimorphic zygomycetes. Accordingly, the present invention relates to a recombinant, fungal cell or dimorphic fungal cell comprising regulatable expression of a regulator of morphol. Expression of the at least one regulator of morpol. directed by the expression signal not natively associated therewith results in a dimorphic shift of dimorphic fungal cell or a desirable, improved filamentation of a fungal cell or a dimorphic fungal cell. The improved filamentation of the fungal cell or the dimorphic fungal cell is pos. correlated with an increased production and/or secretion of desirable polypeptide.

IT **142008-29-5, Protein kinase** A
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(gene pkaR and pkaC for, of Mucor circinelloides; genetic engineering of dimorphic fungi for improved secretion of recombinant proteins)

IT **7647-14-5, Sodium chloride**, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(in culture medium for dimorphic fungi; genetic engineering of dimorphic fungi for improved secretion of recombinant proteins)

L16 ANSWER 10 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 05 Sep 2002
ACCESSION NUMBER: 2002:668948 CAPLUS
DOCUMENT NUMBER: 137:321658
TITLE: cAMP-dependent **protein kinase** inhibits proline transport across the rat renal

tubular brush border membrane
AUTHOR(S): Zelikovic, Israel; Wager-Miller, James
CORPORATE SOURCE: Department of Pediatrics, University of Washington School of Medicine, Seattle, WA, 98105, USA
SOURCE: Bioscience Reports (2001), 21(5), 613-626
CODEN: BRPTDT; ISSN: 0144-8463
PUBLISHER: Kluwer Academic/Plenum Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We studied the effect the cAMP-dependent **protein kinase** (cAK)-mediated phosphorylation on Na+- and Cl--linked proline transport across the rat renal brush border membrane (BBM). CAK bioassay and Western **hybridization** anal. using cAK subunit-specific antibodies demonstrated the presence of the enzyme in the BBM. Brush border membrane vesicles (BBMV) were phosphorylated using the hyposmotic shock technique. CAMP, by activating endogenous cAK, and exogenous, highly purified catalytic subunit of cAK inhibited **NaCl**-dependent proline transport by phosphorylated, lysed/resealed BBMV compared with control vesicles. The cAK-mediated inhibition of proline uptake was completely abolished when phosphorylation at the cytoplasmic (inner side) of the membrane was prevented by isosmotic, rather than hyposmotic, phosphorylation. The cAK-induced inhibition of proline transport was reversed by the specific cAK inhibitor peptide, PK1. These data suggest that cAMP-dependent **protein kinase**-mediated phosphorylation modulates Na+- and Cl--linked proline transport across the tubular luminal membrane.

IT **142008-29-5**, CAMP-dependent **protein kinase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cAMP-dependent **protein kinase** inhibits proline transport across the rat renal tubular brush border membrane)

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 11 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 08 Mar 2002
ACCESSION NUMBER: 2002:172121 CAPLUS
DOCUMENT NUMBER: 136:231255
TITLE: Nucleic acids encoding T-cell activation promoter and cytotoxic agent or cytokine for suppressing or enhancing T cell-mediated immune response
INVENTOR(S): Brenner, Sidney; Venkatesh, Byrappa; Tan, Yin Hwee
PATENT ASSIGNEE(S): Institute of Molecular & Cell Biology, Singapore; Ehrlich, Gal
SOURCE: PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002018619	A2	20020307	WO 2001-IL765	20010816
WO 2002018619	A3	20040715		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ,

LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2419251	AA	20020307	CA 2001-2419251	20010816
AU 2001082458	A5	20020313	AU 2001-82458	20010816
JP 2004520013	T2	20040708	JP 2002-522525	20010816
EP 1456221	A2	20040915	EP 2001-961080	20010816
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2004038247	A1	20040226	US 2003-362010	20030721
PRIORITY APPLN. INFO.:			US 2000-229326P	P 20000901
			WO 2001-IL765	W 20010816

AB An isolated nucleic acid is disclosed, including a promoter sequence being transcriptionally functional in a T-lymphocyte undergoing activation and transcriptionally less functional in the T-lymphocyte prior to the activation. The nucleic acid constructs encode T cell activation promoter sequence and cytotoxic agent for suppressing T cell-mediated immune response and for treating immunol. disorders such as autoimmune diseases. The nucleic acid constructs may encode T cell activation promoter sequence and cytokine for enhancing T cell-mediated immune response and for treating diseases such as viral infection.

IT **7647-14-5, Sodium chloride,** biological studies
RL: ARU (Analytical role, unclassified); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(nucleic acids encoding T-cell activation promoter and cytotoxic agent or cytokine for suppressing or enhancing T cell-mediated immune response)

IT **114051-78-4,** Lck tyrosine kinase
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleic acids encoding T-cell activation promoter and cytotoxic agent or cytokine for suppressing or enhancing T cell-mediated immune response)

L16 ANSWER 12 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 24 May 2001
ACCESSION NUMBER: 2001:374622 CAPLUS
DOCUMENT NUMBER: 135:190312
TITLE: Limbic-cortical-ventral striatal activation during retrieval of a discrete cocaine-associated stimulus: a cellular imaging study with γ **protein kinase** C expression
AUTHOR(S): Thomas, Kerrie L.; Everitt, Barry J.
CORPORATE SOURCE: Department of Experimental Psychology, University of Cambridge, Cambridge, CB2 3EB, UK
SOURCE: Journal of Neuroscience (2001), 21(7), 2526-2535
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We investigated the neuronal activation associated with reexposure to a discrete cocaine-associated stimulus using in situ **hybridization** to quantify the expression of the plasticity-regulated gene, γ **protein kinase** C (γ PKC), in the limbic-cortical-ventral striatal system. Groups of rats were trained to self-administer cocaine paired with a light stimulus (Paired) or paired with an auditory stimulus but also receiving light presentations yoked to those in the Paired group (Unpaired). Addnl. groups received noncontingent cocaine-light pairings (Pavlovian) or **saline**-light pairings (**Saline**) that were yoked to the Paired group. After acquisition of self-administration by the Paired and Unpaired groups, all groups had a 3 d drug- and training-free period before being reexposed to noncontingent presentations of the light conditioning stimulus during a 5 min test session in the training context. There were four major patterns of results for regional γ PKC expression 2 h later. (1) Changes occurred only in groups in which the light was predictive of cocaine. (2) Increases were seen in the amygdala, but decreases were seen in the medial prefrontal cortex. (3) No changes were seen in the hippocampus. (4) Although changes were observed in the basal and central nuclei of the amygdala and the prelimbic cortex in both the Paired and Pavlovian groups, addnl. changes were observed in the nucleus accumbens core, lateral amygdala, and anterior cingulate cortex in the Pavlovian group. These results suggest not only that regionally selective alterations in γ PKC expression are an index of the retrieval of Pavlovian assocns. formed between a drug and a discrete stimulus, but also that a distinct neural circuitry may underlie Pavlovian stimulus-reward assocns. in cocaine-experienced rats.

IT **141436-78-4, Protein kinase** C
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(γ, gene for; limbic-cortical-ventral striatal activation during retrieval of a discrete cocaine-associated stimulus)

REFERENCE COUNT: 86 THERE ARE 86 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 13 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 15 Jan 2001
ACCESSION NUMBER: 2001:32810 CAPLUS
DOCUMENT NUMBER: 134:220187
TITLE: Proline Transport in MDCK Cells Expressing a Mutant Regulatory Subunit of cAMP-Dependent **Protein Kinase**
AUTHOR(S): Zelikovic, Israel; Wager-Miller, James
CORPORATE SOURCE: Department of Pediatrics, Division of Nephrology, University of Washington School of Medicine, Seattle, WA, 98105, USA
SOURCE: Molecular Genetics and Metabolism (2001), 72(1), 45-53
CODEN: MGMEFF; ISSN: 1096-7192
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB CAMP-dependent **protein kinase** (cAK) regulates the activity of several membrane-bound ion channels and carriers. The role of cAK in regulating the transport of osmoprotective amino acids in the distal tubule is unknown. We examined the regulation of Na+- and

Cl--dependent proline transport in MDCK cells expressing a mutant murine regulatory subunit (RIαAB) of cAK. For this purpose, MDCK cells were transfected with an expression vector encoding RIαAB driven by the metallothionein 1 promoter together with the neomycin-resistance (NEO) gene. Stable G418-resistant colonies were isolated that expressed RIαAB as demonstrated by Northern **hybridization** anal. using a cDNA probe for RIα and a cAK assay that showed decreased enzyme activity. A clone constitutively expressing high levels of RIαAB (MAB) in a Zn-independent manner and a control clone transfected with the NEO gene alone (Mneo) were selected for transport studies. We examined the effect of the cAMP-stimulating agents forskolin (F) and IBMX on **NaCl** -dependent uptake of [3H]proline by confluent monolayers of transfected MDCK cells. While F/IBMX-induced mean inhibition of proline transport in Mneo cells was 48 and 45% at 5 and 15 min, resp., inhibition of proline uptake in MAB cells was 9% (5 min) and 0% (15 min). These data demonstrate that the inhibition of **NaCl** -linked proline transport in response to elevated cAMP is reversed in MDCK clones that express mutant cAK and provide evidence that cAK mediates the modulatory action of cAMP on proline transport. CAK may play an important role in controlling transport of proline and other osmoprotective amino acids in the renal tubule. (c) 2001 Academic Press.

IT **142008-29-5**, CAMP-Dependent **protein kinase**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**NaCl**-dependent proline transport in MDCK kidney distal tubule cells expressing mutant regulatory subunit of cAMP-dependent **protein kinase**)

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 14 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 07 Dec 2000
ACCESSION NUMBER: 2000:856693 CAPLUS
DOCUMENT NUMBER: 134:126925
TITLE: A pulmonary rat gene array for screening altered expression profiles in air pollutant-induced lung injury
AUTHOR(S): Nadadur, Srikanth S.; Schladweiler, Mette C. J.; Kodavanti, Urmila P.
CORPORATE SOURCE: Curriculum in Toxicology, University of North Carolina, Chapel Hill, NC, 27711, USA
SOURCE: Inhalation Toxicology (2000), 12(12), 1239-1254
CODEN: INHTE5; ISSN: 0895-8378
PUBLISHER: Taylor & Francis
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Pulmonary tissue injury and repair processes involve complex and coordinated cellular events such as necrosis, inflammation, cell growth/differentiation, apoptosis, and remodeling of extracellular matrix. These processes are regulated by expression of multiple mediator genes. Com. available microarray blots and slides allow screening of hundreds to thousands of genes in a given tissue or cell preparation However, often these blots do not contain cDNAs of one's interest and are difficult to interpret. In order to analyze the tissue expression profile of a large number of genes involved in

pulmonary injury and pathol., the authors developed a rat gene array filter using array technol. This array consisted of 27 genes representing inflammatory and anti-inflammatory cytokines, growth factors, adhesion mols., stress proteins, transcription factors and antioxidant enzymes; 3 neg. controls, and 2 blank spots. Using rat gene-specific polymerase chain reaction (PCR) primer pairs, cDNAs for these genes were amplified and cloned into a TA vector. Plasmids with recombinant cDNA inserts were purified and blotted onto a nylon membrane. Lung total RNA was isolated at 3 or 24 h following intratracheal (IT) exposure of male Sprague Dawley rats to either **saline** (control), residual oil fly ash (ROFA; 3.3 mg/kg) or metals found in one instillate of ROFA: nickel ($NiSO_4$; 1.3 μmol/kg) or vanadium (VSO_4; 2.2 μmol/kg). 12P-Labeled cDNA was generated from RNA samples in a reverse transcriptase reaction and subsequently **hybridized** to array blots. Developing a pulmonary rat gene array may provide a tool for screening the expression profile of tissue specific markers following exposure to toxic air contaminants.

IT **155215-87-5**, Stress activated **protein kinase**
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(gene for; gene array in rat lung for screening altered expression profiles in air pollutant-induced lung injury)

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 15 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 11 Aug 2000
ACCESSION NUMBER: 2000:554583 CAPLUS
DOCUMENT NUMBER: 133:116713
TITLE: Preparation of earthworm kinases
INVENTOR(S): Sun, Qiliang; Feng, Laikun
PATENT ASSIGNEE(S): Gaosida Biochemical Medicines Industrial Group Co., Changchun City, Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 5 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1229852	A	19990929	CN 1999-112806	19990401
CN 1089369	B	20020821		
PRIORITY APPLN. INFO.:			CN 1999-112806	19990401

AB The present invention relates to the preparation of three kinds of earthworm kinasse which have fibrolytic enzyme activities about 1000 units of urokinase. The process mainly comprises chromatog. through GSAC affinity column, ionic exchange DEAE-52 gel, and Sephacryl S-200 column.

IT **994-36-5, Sodium citrate**
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(preparation of earthworm kinases)

L16 ANSWER 16 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN

ED Entered STN: 02 Aug 2000
ACCESSION NUMBER: 2000:525087 CAPLUS
DOCUMENT NUMBER: 134:13858
TITLE: A chromosomally encoded type III secretion pathway in Yersinia enterocolitica is important in virulence
AUTHOR(S): Haller, Jon C.; Carlson, Sharon; Pederson, Kristin J.; Pierson, Dorothy E.
CORPORATE SOURCE: Department of Microbiology, University of Colorado Health Sciences Center, CO, USA
SOURCE: Molecular Microbiology (2000), 36(6), 1436-1446
CODEN: MOMIEE; ISSN: 0950-382X
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Numerous Gram-neg. bacteria use a type III, or contact dependent, secretion system to deliver proteins into the cytosol of host cells. All of these systems identified to date have been shown to have a role in pathogenesis. We have identified 13 genes on the Yersinia enterocolitica chromosome that encode a type III secretion apparatus plus two associated putative regulatory genes. In order to determine the function of this chromosomally-encoded secretion apparatus, we created an in frame deletion of a gene that has homol. to the hypothesized inner membrane pore, ysaV. The ysaV mutant strain failed to secrete eight proteins, called Ysps, normally secreted by the parental strain when grown at 28°C in Luria-Bertani (LB) broth supplemented with 0.4 M **NaCl**. Disruption of the ysaV gene had no effect on motility or phospholipase activity, suggesting this chromosomally encoded type III secretion pathway is distinct from the flagella secretion pathway of Y. enterocolitica. Deletion of the ysaV gene in a virulence plasmid pos. strain had no effect on in vitro secretion of Yops by the plasmid-encoded type III secretion apparatus Secretion of the Ysps was unaffected by the presence or absence of the virulence plasmid, suggesting the chromosomally encoded and plasmid-encoded type III secretion pathways act independently. Y. enterocolitica thus has three type III secretion pathways that appear to act independently. The ysaV mutant strain was somewhat attenuated in virulence compared with the wild type in the mouse oral model of infection (an approx. 0.9 log difference in LD50). The ysaV mutant strain was nearly as virulent as the wild type when inoculated i.p. in the mouse model. A ysaV probe **hybridized** to sequences in other Yersinia spp. and homologues were found in the incomplete Y. pestis genome sequence, indicating a possible role for this system throughout the genus.
REFERENCE COUNT: 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 17 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 30 Jun 2000
ACCESSION NUMBER: 2000:441939 CAPLUS
DOCUMENT NUMBER: 133:69843
TITLE: Cloning and cDNA sequences of a maize **protein kinase** WEE1 homolog involved in cell cycle regulation and uses thereof
INVENTOR(S): Sun, Yuejin; Lowe, Keith S.; Dilkes, Brian R.; Gordon-Kamm, William J.; Larkins, Brian A.; Dante, Ricardo A.
PATENT ASSIGNEE(S): Pioneer Hi-Bred International, Inc., USA; The

	University of Arizona
SOURCE:	PCT Int. Appl., 61 pp.
	CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000037645	A2	20000629	WO 1999-US30957	19991222
WO 2000037645	A3	20001109		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2003041342	A1	20030227	US 1999-470526	19991222
US 6777590	B2	20040817		
PRIORITY APPLN. INFO.:			US 1998-113440P P	19981223

AB The invention provides isolated cDNAs (zmwee1) and their encoded proteins (WEE1) from maize that are involved in cell cycle regulation. The maize WEE1 protein shows 50% identity to the **protein kinase** domain of human Wee1. Zmwee1 transcripts accumulate in maize endosperm during the period of endoreduplication. Overexpression of zmwee1 in Schizosaccharomyces pombe inhibited cell division and caused the cells to enlarge significantly. Recombinant zmwee1 obtained from Escherichia coli is able to inhibit the activity of cyclin-dependent kinase from maize. Zmwee1 is encoded by a single gene at a locus on the long arm of chromosome 4. The invention further provides recombinant expression cassettes, host cells, transgenic plants, and antibody compns. The present invention provides methods and compns. relating to altering cell cycle protein content, cell cycle progression and/or composition of plants.

L16	ANSWER 18 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED	Entered STN: 13 Jan 2000
ACCESSION NUMBER:	2000:31291 CAPLUS
DOCUMENT NUMBER:	132:75412
TITLE:	Cloning of cDNA sequences encoding human **protein kinase** homologs and their potential therapeutic use
INVENTOR(S):	Bandman, Olga; Yang, Y. Tom; Hillman, Jennifer L.; Yue, Henry; Guegler, Karl J.; Corley, Neil C.; Gorgone, Gina A.; Azimzai, Yalda; Lu, Dyung Aina M.
PATENT ASSIGNEE(S):	Incyte Pharmaceuticals, Inc., USA
SOURCE:	U.S., 38 pp.
	CODEN: USXXAM
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6013455	A	20000111	US 1998-173581	19981015
CA 2346775	AA	20000420	CA 1999-2346775	19991015
WO 2000022143	A2	20000420	WO 1999-US24202	19991015
WO 2000022143	A3	20000928		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1121444	A2	20010808	EP 1999-956575	19991015
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002527069	T2	20020827	JP 2000-576033	19991015
US 6264947	B1	20010724	US 1999-420915	19991020
US 2002081290	A1	20020627	US 2001-870962	20010530

PRIORITY APPLN. INFO.:	US 1998-173581	A	19981015
	WO 1999-US24202	W	19991015
	US 1999-420915	A3	19991020

AB The invention provides 9 human **protein kinase** homologs (PKH) and cDNAs which identify and encode PKH. Nucleic acids encoding the kinase homologs were first identified in various Incyte clones using a computer search for amino acid sequence alignments; consensus sequences were derived from overlapping and/or extended nucleic acid sequences. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders associated with expression of PKH.

IT **68-04-2, Trisodium citrate**
7647-14-5, Salt, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(nucleic acid **hybridization;** cloning of cDNA sequences encoding human **protein kinase** homologs and their potential therapeutic use)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 19 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 16 Dec 1999
ACCESSION NUMBER: 1999:794263 CAPLUS
DOCUMENT NUMBER: 132:32682
TITLE: Cloning, sequence and therapeutic use of human **protein kinase** H2LAU20
INVENTOR(S): Brun, Kimberly Anne; Creasy, Caretha Lee; Dunnington, Damien John
PATENT ASSIGNEE(S): SmithKline Beecham Corporation, USA
SOURCE: U.S., 17 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6001623	A	19991214	US 1998-126646	19980731
WO 2000006709	A1	20000210	WO 1999-US16613	19990722
W: JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1100882	A1	20010523	EP 1999-935845	19990722
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002521057	T2	20020716	JP 2000-562491	19990722
US 6365389	B1	20020402	US 1999-421491	19991020

PRIORITY APPLN. INFO.: US 1998-126646 A 19980731
WO 1999-US16613 W 19990722

AB The present invention describes a novel human clone, H2LAU20, which shares homol. with predicted protein serine/threonine kinases from S. pombe, D. melanogaster, C. elegans and S. cerevisiae and has motifs associated with other known **protein kinases.** Inhibitors of H2LAU20 are expected to regulate proliferation of cell growth. The H2LAU20 polypeptides and polynucleotides and methods for producing such polypeptides by recombinant techniques are disclosed. The nucleotide sequence of H2LAU20 shows homol. with human **protein kinase** YAK1 and human **protein kinase** YAK3. The nucleotide sequence of H2LAU20 is a cDNA sequence encoding a polypeptide of 620 amino acids. Also disclosed are methods for utilizing H2LAU20 polypeptides and polynucleotides in therapy, and diagnostic assays for such.

IT **994-36-5, Sodium citrate**
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(SSC (**saline sodium citrate**)-formamide-sodium phosphate buffer; cloning, sequence and therapeutic use of human **protein kinase** H2LAU20)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 20 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 09 Jul 1999
ACCESSION NUMBER: 1999:424998 CAPLUS
DOCUMENT NUMBER: 131:225396
TITLE: Cloning of a novel kinase (SIK) of the SNF1/AMPK family from high salt diet-treated rat adrenal
AUTHOR(S): Wang, Zhi-nong; Takemori, Hiroshi; Halder, Sunil K.; Nonaka, Yasuki; Okamoto, Mitsuhiro
CORPORATE SOURCE: Department of Molecular Physiological Chemistry, Osaka University Medical School, Suita, Osaka, Japan
SOURCE: FEBS Letters (1999), 453(1,2), 135-139
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal

LANGUAGE: English

AB PCR-coupled cDNA subtraction **hybridization** was adapted to identify the genes expressed in the adrenocortical tissues from high salt diet-treated rat. A novel cDNA clone, termed salt-inducible kinase (SIK), encoding a polypeptide (776 amino acids) with significant similarity to protein serine/threonine kinases in the SNF1/AMPK family was isolated. An in vitro kinase assay demonstrated that SIK protein had autophosphorylation activity. Northern blot revealed that SIK mRNA levels were markedly augmented by ACTH treatment both in rat adrenal glands and in Y1 cells. SIK may play an important role in the regulation of adrenocortical functions in response to high plasma salt and ACTH stimulation.

IT **7647-14-5**, Salt, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(cloning of novel kinase (SIK) of SNF1/AMPK family from high salt diet-treated rat adrenal)

IT **243647-13-4**, Salt-inducible kinase
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(cloning of novel kinase (SIK) of SNF1/AMPK family from high salt diet-treated rat adrenal)

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 21 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 27 May 1999
ACCESSION NUMBER: 1999:325803 CAPLUS
DOCUMENT NUMBER: 130:349399
TITLE: High throughput method for functionally classifying proteins identified using a genomics approach
INVENTOR(S): Pantoliano, Michael W.; Salemme, Francis R.; Petrella, Eugenio C.; Carver, Theodore E., Jr.; Rhind, Alexander W.
PATENT ASSIGNEE(S): 3-Dimensional Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9924050	A1	19990520	WO 1998-US24035	19981112
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2309345	AA	19990520	CA 1998-2309345	19981112

AU 9913980	A1	19990531	AU 1999-13980	19981112
AU 750501	B2	20020718		
EP 1030678	A1	20000830	EP 1998-957812	19981112
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2001003648	A1	20010614	US 1998-190128	19981112
JP 2002514571	T2	20020521	JP 2000-520138	19981112
NZ 504483	A	20021126	NZ 1998-504483	19981112
NZ 521789	A	20040227	NZ 1998-521789	19981112
MX 200004638	A	20001110	MX 2000-4638	20000512
US 2002168686	A1	20021114	US 2002-57940	20020129
PRIORITY APPLN. INFO.:			US 1997-65129P	P 19971112
			NZ 1998-504483	A1 19981112
			US 1998-190128	A1 19981112
			WO 1998-US24035	W 19981112

AB The present invention provides a method for functionally classifying a protein that is capable of unfolding due to a thermal change. The method comprises screening one or more of a multiplicity of different mols. for their ability to shift the thermal unfolding curve of the protein, wherein a shift in the thermal unfolding curve indicates that the mol. binds to the protein or affects the stability in a measurable way; generating an activity spectrum for the protein wherein the activity spectrum reflects a set of mols., from the multiplicity of mols., that shift the thermal unfolding curve, of the protein and therefore are ligands that bind to the protein, comparing the activity spectrum for the protein to one or more functional reference spectrum lists; and classifying the protein according to the set of mols. in the multiplicity of different mols. that shift the thermal unfolding curve of the protein. Human Factor Xa and human domain II of the fibroblast growth factor receptor 1 were each assayed by microplate thermal shift assay against a functional library screen in a 96 well plate containing 94 compds. and 2 control wells. The proteins were added to each well along with 1,8-ANS and the microplate reactions were heated simultaneously, in two degree increments, from 40-70°. Fluorescence was measured at 460 nm.

IT **142008-29-5**
RL: BPR (Biological process); BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); BIOL (Biological study); PROC (Process)
(ATP effect on, of bovine heart; high throughput method for functionally classifying proteins identified using genomics approach)

IT **68-04-2, Sodium citrate 7647-14-5**
, Sodium chloride, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(in functional probe library; high throughput method for functionally classifying proteins identified using genomics approach)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 22 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 17 May 1999

ACCESSION NUMBER: 1999:299499 CAPLUS
DOCUMENT NUMBER: 130:322697
TITLE: An in vitro system for inducing neural crest cell differentiation to vascular smooth muscle cells and its use in identifying regulators and their genes
PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA; Lee, Arthur M. E.; Jain, Mukesh; Watanabe, Masafumi
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9921965	A2	19990506	WO 1998-US22897	19981028
WO 9921965	A3	19990826		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9912849	A1	19990517	AU 1999-12849	19981028
EP 1025205	A2	20000809	EP 1998-956294	19981028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001520877	T2	20011106	JP 2000-518057	19981028
PRIORITY APPLN. INFO.:			US 1997-63363P P	19971028
			US 1998-80420P P	19980402
			US 1998-96685P P	19980814
			WO 1998-US22897 W	19981028

AB This invention is directed to an in vitro system for rapidly and uniformly inducing immortalized neural crest cells to differentiate to vascular smooth muscle cells. As excessive proliferation of vascular smooth muscle cells is a phenotypic response to the development of occlusive arteriosclerotic disease, the in vitro system of this invention is used to identify mol. regulators of smooth muscle cell development and differentiation. As the mol. regulators of smooth muscle cell differentiation are identified, the invention also encompasses methods to isolate the genes coding for these regulators. This invention also relates to mols. identified through the use of the invention's in vitro system, as well as to compds. that inhibit or regulate the identified mols. Neural crest Monc-1 cells were induced to differentiate to vascular smooth muscle cells by application of a smooth muscle cell differentiation medium (SMDM) containing inorg. salts, amino acids, vitamins, and other components (specific components are listed) supplemented with 10% fetal bovine serum, penicillin, streptomycin and Hepes (pH 7.4).
IT **172306-54-6**, LIM-kinase 2

RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(as gene product affected, identification of; in vitro system for inducing neural crest cell differentiation to vascular smooth muscle cells and its use in identifying regulators and their genes)

IT **7647-14-5, Sodium chloride**, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(smooth muscle cell differentiation medium containing; in vitro system for inducing neural crest cell differentiation to vascular smooth muscle cells and its use in identifying regulators and their genes)

L16 ANSWER 23 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 08 Apr 1999
ACCESSION NUMBER: 1999:220057 CAPLUS
DOCUMENT NUMBER: 130:247881
TITLE: Plant cyclin-dependent kinase inhibitors and cDNAs and uses for modulation of plant cell growth
INVENTOR(S): Inze, Dirk; De Veylder, Lieven; De Almeida, Janice; Landrieu, Isabelle
PATENT ASSIGNEE(S): CropDesign N.V., Belg.
SOURCE: PCT Int. Appl., 89 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9914331	A2	19990325	WO 1998-EP5895	19980916
WO 9914331	A3	19990610		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2303759	AA	19990325	CA 1998-2303759	19980916
AU 9895406	A1	19990405	AU 1998-95406	19980916
AU 754803	B2	20021128		
EP 1015590	A2	20000705	EP 1998-948981	19980916
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001516582	T2	20011002	JP 2000-511870	19980916
US 6710227	B1	20040323	US 2000-526597	20000316
US 2004073969	A1	20040415	US 2003-688291	20031017

PRIORITY APPLN. INFO.:

EP 1997-202838	A	19970916
EP 1997-204111	A	19971224
WO 1998-EP5895	W	19980916
US 2000-526597	A3	20000316

AB Provided are DNA sequences encoding two A. thaliana and one alfalfa cyclin-dependent kinase Cdc2a inhibitors (CDKI's) as well as methods for obtaining the same. Also disclosed are methods for producing CDKIs with recombinant cells; transgenic plants or plant cells expressing CDKI nucleic acid; regulatory sequences from promoters of the CDKI genes and their use in regulating gene expression in plants; methods for identifying modulators of CDKIs; and uses of CDKI nucleic acids, CDKIs, and antibodies to CDKIs for modulating plant cell cycle and growth, response to environmental stress and male or female sterility. The expression of one of the A. thaliana CDKIs was upregulated by **NaCl**. The A. thaliana CDKI cDNAs were expressed in Schizosaccharomyces pombe. In situ **hybridization** was used to examine CDKI gene expression in various tissues.

IT **143375-65-9**, Cdc2 kinase
RL: AGR (Agricultural use); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process); USES (Uses)
(inhibitors; plant cyclin-dependent kinase inhibitors and cDNAs and uses for modulation of plant cell growth)

L16 ANSWER 24 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 26 Dec 1997
ACCESSION NUMBER: 1997:804769 CAPLUS
DOCUMENT NUMBER: 128:73748
TITLE: Glucose metabolism and insulin receptor binding and mRNA levels in tissues of Dahl hypertensive rats
AUTHOR(S): Sechi, Leonardo A.; Griffin, Chandi A.; Zingaro, Laura; Catena, Cristiana; De Carli, Stefano; Schambelan, Morris; Bartoli, Ettore
CORPORATE SOURCE: Hypertension Unit, Department of Internal Medicine, University of Udine School of Medicine, Udine, 33100, Italy
SOURCE: American Journal of Hypertension (1997), 10(11), 1223-1230
CODEN: AJHYE6; ISSN: 0895-7061
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Increased insulinemic response to an oral glucose load has been demonstrated in Dahl salt-sensitive hypertensive rats. To determine whether this abnormality is mediated at the level of the insulin receptor, the authors compared insulin receptor binding and mRNA levels in tissues of Dahl salt-sensitive rats (DS) and in their normotensive controls, Dahl salt-resistant rats (DR). To evaluate possible influences of dietary sodium intake, rats were fed either low (0.07% **NaCl**) or high salt (7.5% **NaCl**) chow until the DS became hypertensive, and then were killed by decapitation. Fasting plasma glucose and plasma insulin levels did not differ between DR and DS rats and were not affected by salt intake. In response to an oral glucose load, plasma glucose had a similar increase in DR and DS rats, but the increase in plasma insulin was significantly greater in DS rats. Scatchard anal. of binding was obtained from in situ autoradiog. studies performed in frozen skeletal muscle and kidney sections, and insulin receptor mRNA levels were measured by slot-blot **hybridization**. Number and affinity of insulin receptors were comparable in skeletal muscle and kidney of DR and DS rats and, in both groups, binding parameters were not affected

by dietary **sodium chloride**. Hepatic and renal insulin receptor mRNA levels were also comparable in DR and DS rats fed either low or high salt chow. Thus, increased plasma insulin response to oral glucose load is associated with normal insulin receptor binding and gene expression in peripheral tissues in rats with Dahl hypertension. A postreceptor defect is likely responsible for the decreased sensitivity to insulin in this model of genetic hypertension.

IT **7647-14-5, Sodium chloride**, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(increased plasma insulin response to oral glucose load is associated with normal insulin receptor binding and gene expression in peripheral tissues in rats with Dahl hypertension in relation to dietary **sodium chloride**)

IT **88201-45-0**, Insulin receptor tyrosine kinase
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(increased plasma insulin response to oral glucose load is associated with normal insulin receptor binding and gene expression in peripheral tissues in rats with Dahl hypertension in relation to dietary **sodium chloride**)

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 25 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 24 Nov 1997
ACCESSION NUMBER: 1997:736590 CAPLUS
DOCUMENT NUMBER: 128:59746
TITLE: Molecular characterization and in situ localization of a mouse retinal taurine transporter
AUTHOR(S): Vinnakota, Shyamala; Qian, Xiaojun; Egal, Hussein; Sarthy, Vijay; Sarkar, Hemanta K.
CORPORATE SOURCE: Department of Molecular Physiology and Biophysics, Baylor College of Medicine, Houston, TX, USA
SOURCE: Journal of Neurochemistry (1997), 69(6), 2238-2250
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Lippincott-Raven Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Various ocular tissues have a higher concentration of taurine than plasma. This taurine concentration gradient across the cell membrane is maintained by a high-affinity taurine transporter. To understand the physiol. role of the taurine transporter in the retina, the authors cloned a taurine transporter encoding cDNA from a mouse retinal library, determined its biochem. and pharmacol. properties, and identified the specific cellular sites expressing the taurine transporter mRNA. The deduced protein sequence of the mouse retinal taurine transporter (mTAUT) revealed >93% sequence identity to the canine kidney, rat brain, mouse brain, and human placental taurine transporters. Our data suggest that the mTAUT and the mouse brain taurine transporter may be variants of one another. The mTAUT synthetic RNA induced Na+- and Cl--dependent [3H]taurine transport activity in Xenopus laevis oocytes that saturated with an average Km of 13.2 μM for taurine. Unlike the

previous studies, the authors determined the rate of taurine uptake as the external concentration of Cl- was varied, a single saturation process with an average apparent equilibrium constant (KCl-) of 17.7 mM. In contrast, the rate of taurine uptake showed a sigmoidal dependence when the external concentration of Na+ was varied (apparent equilibrium constant, KNa+ .apprx.54.8 mM). Analyses of the Na+- and Cl---concentration dependence data suggest that at least two Na+ and one Cl- are required to transport one taurine mol. via the taurine transporter. Varying the pH of the transport buffer also affected the rate of taurine uptake; the rate showed a min. between pH 6.0 and 6.5 and a maximum between pH 7.5 and 8.0. The taurine transport was inhibited by various inhibitors tested with the following order of potency: hypotaurine > β-alanine > L-diaminopropionic acid > guanidinoethane sulfonate > β-guanidinopropionic acid > chloroquine > γ-aminobutyric acid > 3-amino-1-propanesulfonic acid (homotaurine). Furthermore, the mTAUT activity was not inhibited by the inactive phorbol ester 4α-phorbol 12,13-didecanoate but was inhibited significantly by the active phorbol ester phorbol 12-myristate 13-acetate, which was both concentration and time dependent. The cellular sites expressing the taurine transporter mRNA in the mouse eye, as determined by in situ **hybridization** technique, showed low levels of expression in many of the ocular tissues, specifically the retina and the retinal pigment epithelium. Unexpectedly, the highest expression levels of taurine transporter mRNA were found instead in the ciliary body of the mouse eye.

IT **141436-78-4, Protein kinase** C
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(regulation of mouse retinal taurine transporter by **protein kinase** C)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 26 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 19 Mar 1997
ACCESSION NUMBER: 1997:181106 CAPLUS
DOCUMENT NUMBER: 126:166482
TITLE: Interleukin-1 receptor-associated **protein kinase** and assays
INVENTOR(S): Cao, Zhaodan; Goeddel, David V.; Croston, Glenn E.
PATENT ASSIGNEE(S): Tularik, Inc., USA
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9700690	A1	19970109	WO 1996-US9193	19960605
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5654397	A	19970805	US 1996-587889	19960116
CA 2225450	AA	19970109	CA 1996-2225450	19960605
CA 2225450	C	20010529		

AU 9661766	A1	19970122	AU 1996-61766	19960605
AU 702844	B2	19990304		
EP 839045	A1	19980506	EP 1996-919140	19960605
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 11509085	T2	19990817	JP 1996-503857	19960605
PRIORITY APPLN. INFO.:			US 1995-494006	A 19950623
			WO 1996-US9193	W 19960605

AB The invention relates to human Interleukin-1 Receptor-Associated **Protein Kinases** (IRAKs), nucleic acids which encode IRAKs and **hybridization** probes and primers capable of **hybridizing** with IRAK genes and methods of using the subject compns.; in particular, methods such as IRAK-based in vitro binding assays and phosphorylation assays for screening chemical libraries for lead compds. for pharmacol. agents.

IT **994-36-5, Sodium citrate**
RL: NUU (Other use, unclassified); USES (Uses)
(buffer; interleukin-1 receptor-associated **protein kinase** and assays for screening anti-inflammatory drugs)

L16 ANSWER 27 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 27 Apr 1996
ACCESSION NUMBER: 1996:250476 CAPLUS
DOCUMENT NUMBER: 124:279935
TITLE: Regulation of steady-state concentrations of messenger ribonucleic acid encoding prostaglandin F2α receptor in ovine corpus luteum
AUTHOR(S): Juengel, J. L.; Wiltbank, M. C.; Meberg, B. M.; Niswender, G. D.
CORPORATE SOURCE: Animal Reproduction and Biotechnology Laboratory, Colorado State University, Fort Collins, CO, 80523-1683, USA
SOURCE: Biology of Reproduction (1996), 54(5), 1096-102
CODEN: BIREBV; ISSN: 0006-3363
PUBLISHER: Society for the Study of Reproduction
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To investigate the regulation of ovine luteal receptors for prostaglandin F2α (PGF2α), reverse transcription-polymerase chain reaction was used to produce a 284-bp partial cDNA that was 98% identical to that reported for the bovine PGF2α receptor (PGF2α-R). In situ **hybridization** localized mRNA for PGF2a-R specifically to large luteal cells. In experiment 1, pools of luteal tissue (/day) collected from ewes on Days 3, 6, 9, 12, and 15 of the estrous cycle were analyzed for mRNA encoding PGF2a-R. There was no difference in mean steady-state concns. of mRNA encoding PGF2α-R among any of the days studied (range = 2.3 to 3.5 fmol PGF2α-R mRNA/μg poly[A]+ RNA as assessed by slot-blot **hybridization**). In experiment 2 ewes on Day 11 or Day 12 of the estrous cycle were administered PGF2α ' and corpora lutea were collected 4, 12, or 24 h later (-5 per time point). Nontreated or **saline**-treated ewes served as controls. Luteal concns. of mRNA encoding PGF2α-R were decreased at 4, 12, and 24 h after injection of PGF2α. In experiment 3, ewes (midluteal phase) were administered **saline**, PGF2α, phorbol 12-myristate 13-acetate (PMA), or LH via ovarian arterial injection, and luteal tissue was collected 0, 4, 12, or 24 h later (-4 per treatment per

time). Steady-state concns. of mRNA encoding PGF2α-R were decreased by PGF2α and PMA treatment (4 and 12 h) but were increased at 24 h after LH treatment. In summary, (1) mRNA encoding PGF2α-R was localized to large luteal cells; (2) concns. of mRNA encoding PGF2α-R did not vary during the estrous cycle; (3) treatment with PGF2α or PMA to activate **protein kinase** C decreased concns. of PGF2α-R mRNA within 4 h of treatment; and (4) administration of LH increased concns. of mRNA encoding PGF2α-R 24 h following injection.

IT **141436-78-4, Protein kinase** C
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(regulation of steady-state concns. of mRNA encoding prostaglandin F2α receptor in ovine corpus luteum)

L16 ANSWER 28 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 12 May 1995
ACCESSION NUMBER: 1995:543568 CAPLUS
DOCUMENT NUMBER: 122:285539
TITLE: A serine/threonine **protein kinase** that phosphorylates the N-terminal activation domain of the c-jun protein
INVENTOR(S): Karin, Michael; Davis, Roger; Hibi, Masahiko; Lin, Anning; Derijard, Benoit
PATENT ASSIGNEE(S): University of California, USA; University of Massachusetts
SOURCE: PCT Int. Appl., 142 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9503323	A1	19950202	WO 1994-US8119	19940718
W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LT, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5534426	A	19960709	US 1993-94533	19930719
US 6514745	B1	20030204	US 1994-220602	19940325
AU 9473380	A1	19950220	AU 1994-73380	19940718
AU 700137	B2	19981224		
EP 726908	A1	19960821	EP 1994-923544	19940718
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09507384	T2	19970729	JP 1995-505262	19940718
JP 2925740	B2	19990728		
CA 2166981	C	20001107	CA 1994-2166981	19940718

PRIORITY APPLN. INFO.:

US 1993-94533	A	19930719
US 1994-220602	A	19940325
WO 1994-US8119	W	19940718

AB An isolated 46 kDa (by reducing SDS-PAGE) protein (JNK) with a serine/

threonine kinase activity that phosphorylates the c-Jun N-terminal activation domain and methods of detecting the protein are described. CDNAs encoding the protein are also described. JNK phosphorylates c-Jun N-terminal activation domain which affects gene expression from AP-1 sites. Proteins binding c-jun were identified by affinity chromatog. against immobilized c-jun and a c-jun kinase activity was detected and characterized. The binding of the kinase to c-jun was strong with most of the complex stable to **NaCl** 2M. The roles of the protein in c-jun activation, its role in the interaction of c-jun and c-Ha-ras proteins and in T-cell activation are studied.

IT **160081-84-5**
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(amino acid sequence; serine/threonine **protein kinase** that phosphorylates N-terminal activation domain of c-jun protein)

IT **155215-87-5**
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(serine/threonine **protein kinase** that phosphorylates N-terminal activation domain of c-jun protein)

L16 ANSWER 29 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 24 Dec 1994
ACCESSION NUMBER: 1995:260482 CAPLUS
DOCUMENT NUMBER: 122:126611
TITLE: A **NaCl**-regulated plant gene encoding a brain protein homolog that activates ADP ribosyltransferase and inhibits **protein kinase** C
AUTHOR(S): Chen, Zutang; Fu, Haian; Liu, Dong; Chang, Pi-Fang Linda; Narasimhan, Meena; Ferl, Robert; Hasegawa, Paul M.; Bressan, Ray A.
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02111, USA
SOURCE: Plant Journal (1994), 6(5), 729-40
CODEN: PLJUED; ISSN: 0960-7412
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A cDNA clone pCZ1, with a 1.1 kb insert, was isolated from a **NaCl**-adapted tobacco cell cDNA library that encodes an apparently full-length 29 kDa protein (251 amino acids) with a calculated pI of 5.7. The encoded peptide had a high amino acid sequence identity with bovine 14-3-3 protein which was originally found as an abundant protein in the animal central nervous system. Recently, proteins with sequence identity to 14-3-3 protein have also been found in plants, insects and yeast, and appear to have diverse physiol. functions. Similar to the bovine brain 14-3-3 protein, the recombinant pCZ1 protein stimulated ADP-ribosylation of protein substrate by ADP-ribosyl-transferase from the plant and animal pathogenic bacterium Pseudomonas aeruginosa. This recombinant protein also inhibited **protein kinase** C activity in vitro. Southern blot analyses indicated that most likely five genes encoding 14-3-3-like proteins are present in tobacco. The pCZ1 cDNA insert **hybridized** to a single mRNA of 1.1 kb from cultured tobacco cells. The level of this mRNA transcript in tobacco cells was down-regulated upon adaptation to **NaCl** but was unaffected by

short-term treatment with **NaCl**, ABA or ethylene. In tobacco plants, expression of transcript that **hybridized** to pCZ1 was tissue specific, and was most abundant in roots and flower parts. Monoclonal antibody raised against GF14 protein, a maize protein with substantial sequence identity with 14-3-3 protein detected two bands on SDS-PAGE of total proteins from unadapted tobacco cells and only a single band from cells adapted to **NaCl**. The GF14 antibody was also used to illustrate that the G-box element of a salt-induced gene is associated with a 14-3-3-type protein.

IT **141436-78-4, Protein kinase** C
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(inhibition of, by tobacco **protein kinase**; sequence and characterization of a **NaCl**-regulated tobacco gene encoding protein 14-3-3 which activates ADP ribosyltransferase and inhibits **protein kinase** C)

IT **7647-14-5, Sodium chloride (NaCl)**, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(sequence and characterization of a **NaCl**-regulated tobacco gene encoding protein 14-3-3 which activates ADP ribosyltransferase and inhibits **protein kinase** C)

L16 ANSWER 30 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 14 May 1993
ACCESSION NUMBER: 1993:186082 CAPLUS
DOCUMENT NUMBER: 118:186082
TITLE: Cloning and sequencing of a rabbit cDNA encoding an intestinal and kidney-specific sodium/hydrogen ion exchanger isoform (NHE-3)
AUTHOR(S): Tse, Chung Ming; Brant, Steven R.; Walker, M. Susan; Pouyssegur, Jacques; Donowitz, Mark
CORPORATE SOURCE: Sch. Med., Johns Hopkins Univ., Baltimore, MD, 21205, USA
SOURCE: Journal of Biological Chemistry (1992), 267(13), 9340-6
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Two distinct mammalian Na+/H+ exchanger isoforms (NHE-1 and NHE-2) were previously cloned, sequenced, and expressed. The cloning of a composite cDNA which encodes a third mammalian isoform (NHE-3), which is expressed specifically in intestine and kidney is reported here. The protein deduced from the longest open reading frame of this composite sequence has 832 amino acids with a calculated Mr of 92,747. The hydrophobicity plot of NHE-3 is very similar to that of NHE-1 and NHE-2. NHE-3 is also predicted to have 10-12 membrane-spanning domains and a long cytoplasmic domain which contains putative **protein kinase** phosphorylation motifs. NHE-3 exhibits overall 41% amino acid identity with NHE-1. NHE-3 is likely a glycoprotein as it has one potential N-linked glycosylation site, which is conserved in all NHEs identified. Northern blot anal. of poly(A+) RNA isolated from rabbit ileum using NHE-3 cDNA as a probe **hybridized** to a single 5.4-kilobase transcript. More detailed tissue distribution of message was performed by RNase protection assay. It was found that NHE-3 message is only expressed in intestine and kidney, with the kidney cortex having the most abundant message,

followed by intestine and kidney medulla. In intestine, ileum and ascending colon have the same amount of message, with much lesser amts. in jejunum. The message is absent from duodenum and descending colon, which lack the neutral **NaCl** absorptive process. Thus, NHE-3 might be involved in Na+ absorption in intestinal and renal epithelial cells.

L16 ANSWER 31 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 04 Feb 1990
ACCESSION NUMBER: 1990:33761 CAPLUS
DOCUMENT NUMBER: 112:33761
TITLE: Levels of transcripts encoding a member of the **protein kinase** C family in the paraventricular and supraoptic nuclei are increased by hyperosmolality
AUTHOR(S): Young, W. Scott, III
CORPORATE SOURCE: Lab. Cell Biol., Bethesda, MD, 20892, USA
SOURCE: Journal of Neuroendocrinology (1989), 1(2), 79-82
CODEN: JOUNE2; ISSN: 0953-8194
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The distribution of mRNA transcripts for the 6 known **protein kinase** C (PCK) genes was investigated by quant. in situ **hybridization** histochem. PKC-δ transcripts were detected only in the thalamus, and PKC-ζ was found in several areas including the supraoptic (SON) and paraventricular nuclei (PVN). Activation of magnocellular cells in the SON and PVN following 7 days of **saline** loading resulted in increased levels of PKC-ζ transcripts in both of these nuclei.
IT **7647-14-5, Sodium chloride**, biological studies
RL: BIOL (Biological study)
(**protein kinase** C isoenzyme-specific mRNAs in brain hypothalamus regulation by)

L16 ANSWER 32 OF 32 CAPLUS COPYRIGHT 2006 ACS on STN
ED Entered STN: 12 May 1984
ACCESSION NUMBER: 1983:466219 CAPLUS
DOCUMENT NUMBER: 99:66219
TITLE: Characterization of a messenger RNA transport protein
AUTHOR(S): Moffett, R. Bruce; Webb, Thomas E.
CORPORATE SOURCE: Coll. Med., Ohio State Univ., Columbus, OH, 43210, USA
SOURCE: Biochimica et Biophysica Acta, Gene Structure and Expression (1983), 740(3), 231-42
CODEN: BBGSD5; ISSN: 0167-4781
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A cytoplasmic protein which facilitates the energy-dependent transport of mRNA from isolated nuclei to a specified medium was characterized. This protein was shown, by cDNA **hybridization** anal. with appropriate recombinant probes, to be obligatory for the transport of α2u-globulin and albumin mRNA from male rat liver nuclei. It is concentrated in the cytoplasm. When isolated under conditions where they retain nuclear proteins, the nuclei contain <2% of the total mRNA transport activity. Approx. 20% is recovered in the cytosol, whereas the rest (80%) copurifies with the messenger ribonucleoproteins in the polyribosome fraction. The protein is eluted from the

poly(A)-messenger ribonucleoproteins between 0.25 and 0.50 M **NaCl**. The activities of the cytosolic- and messenger ribonucleoprotein-derived transport proteins were mutually additive below saturation of the transport system. Further, the activities of both fractions were increased when they were fortified with the catalytic subunit of the cAMP-dependent **protein kinase** in the presence of ATP. However, **protein kinase** -induced thiophosphorylation of the protein with ATP[S] decreased transport activity. The mol. weight of the transport protein from either cell compartment is .apprx.35,000. It was purified 2000-fold and requires Mn2+ and serum albumin for stabilization of activity. The highly purified transport factor from the cytosol is tentatively assigned a mol. weight of 32,000 by SDS-polyacrylamide gel electrophoresis.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 14:08:07 ON 14 MAR 2006)

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L17          16 SEA ABB=ON  PLU=ON  L12
L18        3927 SEA ABB=ON  PLU=ON  L8(L)(L9 OR L10)
L19          52 SEA ABB=ON  PLU=ON  L18(L)(HYBRIDIS? OR HYBRIDIZ?)
L20          52 SEA ABB=ON  PLU=ON  L19(L)(POLYPEPTIDE OR PEPTIDE OR
                PROTEIN OR POLYPROTEIN)
L21          59 SEA ABB=ON  PLU=ON  L17 OR L20
L22          39 DUP REM L21 (20 DUPLICATES REMOVED)
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L22 ANSWER 1 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2006-079834 [08] WPIDS
DOC. NO. NON-CPI: N2006-069129
DOC. NO. CPI: C2006-028911
TITLE: Determining subject having or predisposed for mood disorder comprises adding reagent which selectively associates with polynucleotide or polypeptide encoded by hybridizing sequence for gene such as chimerin, ribophorin II, and villin II.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): AKIL, H; BUNNEY, W E; CHOUDARY, P V; EVANS, S J; JONES, E G; LI, J; LOPEZ, J F; LYONS, D M; MOLNAR, M; MYERS, R M; SCHATZBERG, A F; STEIN, R; THOMPSON, R C; TOMITA, H; VAWTER, M P; WATSON, S J
PATENT ASSIGNEE(S): (STRD) UNIV LELAND STANFORD JUNIOR
COUNTRY COUNT: 111
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2006002262	A2	20060105	(200608)*	EN	246

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 2006002262 A2 WO 2005-US22111 20050621

PRIORITY APPLN. INFO: US 2005-667296P 20050331; US 2004-581998P 20040621; US 2004-621252P 20041022

AN 2006-079834 [08] WPIDS

AB WO2006002262 A UPAB: 20060201

NOVELTY - Determining (M1) whether a subject has or is predisposed for mood disorder, involves contacting subject's biological sample with reagent that selectively associates with a polynucleotide or **polypeptide** encoded by a nucleic acid that **hybridizes** under stringent conditions to sequence (NS) of genes associated with mood disorder (e.g. chimerin, ribophorin II, and villin II); and detecting level of reagent that selectively associates with sample.

DETAILED DESCRIPTION - Determining (M1) whether a subject has or is predisposed for a mood disorder, involves (a) obtaining a biological sample from a subject; contacting the sample with a reagent that selectively associates with a polynucleotide or **polypeptide** encoded by a nucleic acid that **hybridizes** under stringent conditions to a nucleotide sequence (NS) of genes associated with bipolar disorder (BP) and major depressive disorder (MDD) chosen from genes associated with BP (e.g. chimerin and ribophorin II), genes associated with MDD (e.g. villin II and pleiotrophin), growth factor pathway related genes (e.g. epidermal growth factor receptor pathway substrate 8, and fibroblast growth factor 12), and G **protein**-coupled receptor (GPCR) pathway genes (e.g. neuropeptide Y, guanine nucleotide binding **protein** and mitogen-activated **protein kinase** 1) given in the specification; and detecting the level of reagent that selectively associates with the sample, thus determining whether the subject has or is predisposed for a mood disorder, (b) contacting the tissue of one or more regions of the subject's brain with a detectably labeled molecule that selectively binds to gene (G1) chosen from NS, BP and MDD associated genes such as gamma -aminobutyric acid (GABA)/glutamate signalling genes (e.g. GABA receptor alpha 5 and glutamate receptor metabotropic 3, genes of G **protein** and GPCR signalling pathways (e.g. cholecystokinin and cyclin-dependent kinase 5), mitochondrial genes (e.g. chromosome 14 open reading frame 2, vesicle-associated membrane **protein** 1, and mitochondrial folate transporter), genes dysregulated in MDD, BP and schizophrenia (e.g. kelch-like 12, alpha -2-macroglobulin, and prostaglandin), GPCR and related signalling genes dysregulated in anterior cingulated cortex, dorsolateral prefrontal cortex and cerebellar cortex (e.g. neurotensin receptor 2, G **protein** beta 5 and proenkephalin), GPCRs and related signalling genes dysregulated in amygdala, hippocampus and nucleus accumbens of MDD and BPD (e.g. adrenomedullin and serotonin receptor), mitochondria, chaperone, apoptosis and proteasome associated genes dysregulated in BPD or MDD (e.g. catalase and heat shock 70 kDa **protein** 2), mitochondrial related genes (e.g. cytochrome c oxidase, subunit 1), genes being upregulated and downregulated by lithium in monkey brains (e.g. zinc finger **protein**, multitype 1, and HSPC142 **protein**), and genes differentially expressed in frontal cortex of rats subjected to chronic unpredictable stress (e.g. calpail, small subunit), given in the specification; visualizing the distribution of the detectably labeled molecule in the brain tissue; and correlating the distribution of the detectably labeled molecule with the presence of or predisposition for a mood disorder in the subject, or (c) obtaining a

biological sample from a subject; contacting the sample with a reagent that selectively associates with a polynucleotide or **polypeptide** encoded by a nucleic acid that **hybridizes** under stringent conditions to a nucleotide sequence of mitochondrial genes expressed in MDD and BP, GPCRs and related signalling genes dysregulated in amygdala, hippocampus and nucleus accumbens of MDD or BPD, genes associated with mitochondria, chaperone, apoptosis and proteasome, and genes differentially expressed in frontal cortex of rats subjected to chronic unpredictable stress; and detecting the level of reagent that selectively associates with the sample, thus determining whether the subject has or is predisposed for a mood disorder.

INDEPENDENT CLAIMS are also included for the following:

(1) identifying (M2) a compound for treatment or prevention of a mood disorder;

(2) treating (M3) a mood disorder in a subject; and

(3) determining (M4) the course of progression or regression of a mood disorder.

ACTIVITY - Neuroleptic; Antidepressant; Tranquilizer. In vivo analysis of the fibroblast growth factor (FGF)-2 in altering hippocampal volume, emotional reactivity and learning and memory was carried as follows. Sprague-Dawley rats were injected with either vehicle or FGF-2 (20 ng/g) on postnatal day 2 (PD2). Three weeks after injection, the dentate gyrus volume and cell counts were evaluated by Nissl staining. The neurogenesis was evaluated by BrdU and Ki-67 immunohistochemistry at the 23 day time point. In adult rats, the locomotor activity, anxiety behaviour and learning and memory, were also evaluated. Results indicated that the FGF-2 injected rats exhibited a 10.5% increase in dentate gyrus volume. The results showed that FGF-2 significantly increased locomotor activity over controls in a novel environment. Increased activity in response to novelty has been associated with a host of other measures including decreased anxiety-like behaviour.

MECHANISM OF ACTION - Phosphoserine phosphatase-like modulator; FGF agonist.

USE - (M1) is useful for determining whether a subject has or is predisposed for a mood disorder, where the mood disorder is chosen from bipolar disorder, major depressive disorder and chronic stress. (M3) is useful for treating a mood disorder in a subject (claimed). (M1) is useful for determining a patient suffering from disorders such as psychotic-depression, depression and anxiety, melancholic depression, schizophrenia and chronic depression.

DESCRIPTION OF DRAWING(S) - The figure is a graph representing the mean grayscale (n=6/group) intensity and standard error bars for 35 S signal for regions indicated for both fluoxetine treated and **saline** treated rats.

Dwg.1/31

L22 ANSWER 2 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2006-088671 [09] WPIDS
CROSS REFERENCE: 2003-268105 [26]; 2004-022840 [02]
DOC. NO. CPI: C2006-031986
TITLE: New antisense oligonucleotides useful for shifting liver gene expression profile of obese animal to profile of lean animal are targeted to a nucleic acid encoding apolipoprotein B.
DERWENT CLASS: B04 D16
INVENTOR(S): CROOKE, R M; GRAHAM, M J
PATENT ASSIGNEE(S): (CROO-I) CROOKE R M; (GRAH-I) GRAHAM M J

COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2006009410	A1	20060112	(200609)*		56

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2006009410	A1 CIP of	US 2003-712795	20031113
	Provisional	US 2004-568825P	20040505
		US 2005-123656	20050505

PRIORITY APPLN. INFO: US 2004-568825P 20040505; US 2003-712795 20031113; US 2005-123656 20050505

AN 2006-088671 [09] WPIDS
CR 2003-268105 [26]; 2004-022840 [02]
AB US2006009410 A UPAB: 20060206

NOVELTY - Antisense oligonucleotides (I) containing 15 - 30 nucleobases and targeted to a nucleic acid encoding apolipoprotein B, are new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for modulating the level of apolipoprotein B gene target mRNA selected from Lcat, Lip1, LipC, Ppara, Pprag, Pcx, Apoa4, Apoc1, Apoc2, Apoc4, Mttp, Prkaa1, Prkaa2, Prkab1, Prkag1, Strebp-1, Scd2, Scd1, Acadl, Acadm, Acds, Acox1, Cpt1a, Cpt2, Crat, Elov12, Elov13, Acadsb, Fads2, Fasn, Facl2, Facl4, Abcd2, Dbi, Fabp1, Fabp2, Fabp7, Acta-1, Acca-1, Cyp7a1, Cyp7b1, Soat2, Ldlr, Hmgcs2, Car5a, Gck or G6 pc involving: contacting an animal with (I).

ACTIVITY - Anorectic; Cardiovascular-Gen.; Antilipemic; Cytostatic.

An antisense oligonucleotide ISIS 147754 (a) was tested for lean animal characteristic by measuring AMP-activated **protein kinase** (AMPK); a sensor for glucose and lipid metabolism. Male C57B1/6 mice on high fat diet were fed with (a) (10, 25 and 50 mg/kg) twice weekly. The control comprised untreated high fat diet fed mice. The (%) expression of AMPK- alpha 2 was found to be increased by 41, 49 and 87, in mice treated with (a) (10, 25 and 50 mg/kg), respectively.

MECHANISM OF ACTION - Liver gene expression profile modulator; Apolipoprotein B mRNA modulator; Breast cancer cell apoptosis inducer; Angiogenesis inhibitor; Leptin secretion enhancer; T-cell co-stimulation inhibitor.

An antisense oligonucleotide having a sequence gtccctgaagatgtcaatgc (a) was tested for modulation of apolipoprotein B mRNA levels and gene expression pattern, by DNA array analysis. Male C57B1/6 mice on high fat diet were injected with (a) (25 mg/kg) twice weekly for 6 weeks. The controls comprised mice on lean diet and injected with **saline**, and mice on high fat diet and injected with **saline**. Comparative mice were fed on high fat diet and given atorvastatin calcium (20 mg/kg) daily for 6 weeks, or treated with a control oligonucleotide having sequence ccttccctgaaggttcctcc (b) that does not target apolipoprotein B. The apolipoprotein B mRNA expression was measured by real-time PCR. The % expression of genes normalized to high fat diet, **saline** treated mice in treated

with (a)/**saline**/(b)/atorvastatin were: ABCD2 = 5/193/32/69; SCD1 = 4/64/43/25; HMGCR = 102/117/131/358; and FABP2 = 32/28/109/63.

USE - The antisense molecule is used for modulating the level of apolipoprotein B gene target mRNA, useful shifting a liver gene expression profile of an obese animal to that of a lean animal, for lowering high cardiovascular risk profile defined by ATPIII, and for inducing apoptosis of cancer cells, such as breast cancer cells (claimed).

ADVANTAGE - The antisense oligonucleotides modulate several apolipoprotein B mRNAs and reduce the level of apolipoprotein B by specifically **hybridizing** to and inhibiting the expression of nucleic acid molecule encoding apolipoprotein B. Thus lower serum cholesterol levels. The oligonucleotides reduce the apolipoprotein B mRNA in a time dependent and dose dependent manner. The modulation results in a shift of a live gene expression profile of an obese animal to that of a lean animal, by altering several cellular pathways or metabolic processes of apoptosis, angiogenesis, leptin secretion and T-cell co-stimulation, such as inhibit angiogenesis, increase leptin secretion and inhibit T-Cell co-stimulation.

Dwg.0/0

L22 ANSWER 3 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2006-056703 [06] WPIDS
DOC. NO. CPI: C2006-021247
TITLE: Pharmaceutical composition useful for treating cognitive deficit associated with, e.g. Down's syndrome comprises hydroxymethylglutaryl Co-enzyme A reductase inhibitor.
DERWENT CLASS: B05
INVENTOR(S): SILVA, A J
PATENT ASSIGNEE(S): (REGC) UNIV CALIFORNIA
COUNTRY COUNT: 111
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2005120496	A2	20051222	(200606)*	EN	66

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005120496	A2	WO 2005-US18129	20050523

PRIORITY APPLN. INFO: US 2005-661764P 20050314; US 2004-574442P 20040524

AN 2006-056703 [06] WPIDS

AB WO2005120496 A UPAB: 20060124

NOVELTY - A pharmaceutical composition comprising an hydroxymethylglutaryl Co-enzyme A (HMG-CoA) reductase inhibitor,

excipient and optionally other inhibitor selected from farnesyl transferase inhibitor, geranylgeranyltransferase inhibitor and an inhibitor of gamma aminobutyric acid (GABA) receptor activity, where the inhibitor of GABA receptor activity is selective for GABA-A/B and the HMG CoA inhibitor does not significantly lower serum cholesterol level, is new.

ACTIVITY - Nootropic; Tranquilizer.

Efficacy of lovostatin (Ia) was tested for treating learning deficit in C57BL/6N type mice of neurofibromatosis-1. The mice treated with (Ia) (0 - 50 mg/kg) were isolated and homogenized in **protein** extraction buffer, with 1% Triton X-100, HEPES (25 mM) (pH of 7.5), **NaCl** (150 mM), 10% glycerol EDTA (2 mM), 1 ug mg-1 leupeptin, 100 ug ml-1 PMSF, NaF (10 mM),.Na glycerophosphate (25 mM) and Na3VO4 (1 mM). Supernatant was collected after 10 minutes of 13000 revolutions per minute centrifugation. **Protein** concentrations were determined by bicirchoninic acid **protein** assay. Lysates were added to SDS loading buffer and boiled 2 minutes. Products were separated by electrophoresis. Gels were blotted to nitrocellulose membrane, blocked for 1 hour at room temperature, washed in tris-buffered **saline** and **hybridized** for 1 hour at room temperature with anti-phospho-p44/42 antibody diluted in TBS, 0.1% Tween 20 (RTM) and non fat dry milk (5 weight/volume%). The membranes were processed and stripped in strip buffer at room temperature for 15 minutes. The results showed that the dose was effective at ameliorating the abnormally high p21Ras/MAPK activity in the mice.

MECHANISM OF ACTION - Hydroxymethylglutaryl Co-enzyme A (HMG-CoA) reductase inhibitor.

USE - For treating cognitive deficit associated with (e.g. Down's syndrome, Angelman syndrome, NF-1 genetic defect, genetic defect in OPHN1, tuberous sclerosis, autism, attention deficit/hyperactivity disorder, dysregulation of small monomeric GTP binding **protein** activity (such as RAS **protein** associated with NF-1 genetic effect), dysregulation of mitogen activated **protein** **kinase** (MAPK) signaling pathway, increased inhibitory neuronal activity, increased GABA-mediated inhibition and activity of GABA-A/B) in a subject with normal cholesterol level; and for modulating long term potentiation (LTP) in a neutral system (such as CA1 region of hippocampus) with a depressed LTP response associated with the dysregulation of small monomeric GTP binding **protein** activity, MAPK signaling pathway or inhibitory neuronal activity that is associated with increased GABA-mediated inhibition (claimed). Also useful for treating memory and learning deficits.

ADVANTAGE - The HMG CoA inhibitor is present in an amount that does not significantly lower serum cholesterol level; hence effective in treating cognitive disorder associated with Rap **protein** and regulates MAPK pathway.

Dwg.0/4

L22 ANSWER 4 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER:	2005-091483 [10] WPIDS
DOC. NO. CPI:	C2005-030849
TITLE:	New 5-substituted-(4-substituted phenylamino)-2-pyridone derivatives useful in the treatment of e.g. proliferative diseases, cancer, autoimmune disease, atherosclerosis and chronic pain.
DERWENT CLASS:	B02 B03
INVENTOR(S):	BLACK, S L; KAUFMAN, M D; ORTWINE, D F; PLUMMER, M S; QUIN, J; REWCASTLE, G W; SHAHRIPOUR, A B; SPICER, J

A; WHITEHEAD, C E
PATENT ASSIGNEE(S): (WARN) WARNER LAMBERT CO LLC
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2005000818	A1	20050106	(200510)*	EN	136

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

US 2005026964 A1 20050203 (200511)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005000818	A1	WO 2004-IB2060	20040618
US 2005026964	A1 Provisional	US 2003-483307P	20030627
	Provisional	US 2003-489603P	20030723
		US 2004-876100	20040624

PRIORITY APPLN. INFO: US 2003-489603P 20030723; US 2003-483307P 20030627; US 2004-876100 20040624

AN 2005-091483 [10] WPIDS

AB WO2005000818 A UPAB: 20050211

NOVELTY - 5-Substituted-(4-substituted phenylamino)-2-pyridone derivatives or their salts, 1-6C amides or 1-6C esters are new.

DETAILED DESCRIPTION - 5-Substituted-(4-substituted phenylamino)-2-pyridone derivatives of formula (I) or their salts, 1-6C amides or 1-6C esters are new.

W = -COOH, -COQ, (1,3,4)oxadiazol-2-yl (substituted on 5-position by Z) or (1,3,4)-oxadiazol-2-one-5-yl;

Q = NH2 (optionally mono or di-substituted by methyl or amino), O-(CH2)k-CH3, NH((CH2)kCH3) or NH(O(CH2)kCH3) (where the (CH2)kCH3 moieties of O-(CH2)kCH3, NH(CH2)kCH3 and NH(O(CH2)kCH3) are optionally mono- - tri-substituted by OH, amino or (cyclo)alkyl);

Z = NH2 (optionally mono or di-substituted by methyl or amino), NH((CH2)kCH3) or NH(O(CH2)kCH3) (where the (CH2)kCH3 moieties of NH(CH2)kCH3 and NH(O(CH2)kCH3) are optionally mono- - tri-substituted by OH or amino);

R1 = 1-6C alkyl (optionally mono or di-substituted by OH, COOH or CN), 2-4C alkenyl, (CH2)kO(CH2)kOCH3 or H;

R2 = H, Cl, F or CH3;

R3 = R2 or CF3;

R4 = 1-6C alkyl, 2-6C alkynyl (both optionally mono - tri-substituted by OH or alkyl), Br, Cl, F, I, 2-4C alkenyl, 3-6C cycloalkyl, (CH2)-3-6C cycloalkyl, CN, O-1-4C alkyl, S-1-2C alkyl, SOCH3, SO2CH3, SO2NR6R7, C triple bond C-(CH2)nNH2, C triple bond C-(CH2)nNHCH3, C triple bond C-(CH2)nN(CH3)2, C triple bond C-CH2OCH3, C=C-(CH2)nOH, C=C-(CH2)nNH2, (Z)-CHCHCH2OCH3, (Z)-CHCH(CH2)nNHCH3, (Z)-CHCH(CH2)nN(CH3)2, (CH2)pCO2R6, CO-1-3C alkyl, C(O)NHCH3,

(CH2)mNH2, (CH2)mNHCH3, (CH2)mN(CH3)2, (CH2)mOR8, CH2S(CH2)t(CH3), (CH2)pCF3, C triple bond C-CF3, CH=CH-CF3, CH2CHCF2, CH=CF2, (CF2)vCF3, CH2(CF2)nCF3, (CH2)tCF(CF3)2, CH(CF3)2, CF2CF(CF3)2 or C(CF3)3;

R3+R4 = 6-membered aryl ring, 5-membered cycloalkyl ring or 5 or 6-membered heteroaryl ring;

R5 = H, Cl, F or CH3;

R6 and R7 = H, methyl or ethyl;

k = 0 - 3;
m = 1 - 4;
n = 1 or 2;
p = 0 - 2;
t = 0 or 1;
v = 1 - 5.

An INDEPENDENT CLAIM is included for use of (I) in combination with radiation therapy or at least one chemotherapeutic agent for the treatment of cancer.

ACTIVITY - Cytostatic; Vasotropic; Antipsoriatic; Immunosuppressive; Antiarteriosclerotic; Antirheumatic; Antiarthritic; Cardiant; Analgesic; Osteopathic; Antidiabetic; Nephrotropic; Ophthalmological; Antibacterial; Virucide; Hepatotropic; Cerebroprotective; CNS-Gen.; Respiratory-Gen.; Antiasthmatic. Test details described but no results given.

MECHANISM OF ACTION - Cancer cell growth inhibitor; Mitogen activated **protein kinase** (MAPK)/extracellular signal regulated kinase (ERK) (i.e. MEK) inhibitors.

4-(2-Fluoro-4-iodoanilino)-N-(2-hydroxyethoxy)-1-methyl-6-oxo-1,6-dihydro-3-pyridinecarboxamide (Ia) was tested for MEK inhibitory activity by determining its ability to inhibit phosphorylation of MAP kinase (extracellular signal regulated kinase (ERK)) in murine colon 26 carcinoma cells. The detection was carried out by Western blot or ELSA format. For Western blot, cells were rinsed free of (A)/vehicle and lysed in a solution containing **NaCl** (70 mM), glycerol phosphate (50 mM), HEPES (10 mM), pH 7.4, 1% Triton X-100 (RTM), Na3VO4 (1 mM), PMSF (100 micro M), leupeptin (10 micro M) and pepstatin (10 micro M). Supernatants were subjected to gel electrophoresis and **hybridized** to primary recognizing dually phosphorylated ERK1 and ERK2. Blots were stripped and re-probed with 1:1 mixture of polyclonal antibodies recognizing un-phosphorylated ERK1 and ERK2. C26 cells were treated with (A) or vehicle for 1 hour at 37 deg. C. (Ia) Showed an IC50 of 0.0438 mM.

USE - In the preparation of composition for the treatment of proliferative diseases, cancer, restenosis, psoriasis, autoimmune disease, atherosclerosis, rheumatoid arthritis, heart failure, chronic pain, neuropathic pain and osteoarthritis (claimed); for treating diseases related to the hyperactivity of MEK and diseases modulated by MEK cascade; also in the treatment of stroke, septic shock, tumors, cystic fibrosis, complications of diabetes, diabetic retinopathy, diabetic nephropathy, hepatomegaly, cardiomegaly, acute focal ischemic stroke, septic shock, asthma, Alzheimer's diseases; as antiviral agent for treating viral infections such as HIV, hepatitis B virus, cytomegalovirus, and Epstein-Barr virus.

ADVANTAGE - The compounds are potent mitogen activated **protein kinase** (MAPK)/extracellular signal regulated kinase (ERK) i.e. MEK kinase inhibitors.

Dwg.0/0

L22 ANSWER 5 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-099932 [11] WPIDS

CROSS REFERENCE: 1999-045179 [04]; 2001-226663 [23]; 2002-024658 [03]; 2003-874615 [81]; 2004-031296 [03]; 2005-072236 [08]; 2006-016984 [02]
DOC. NO. CPI: C2005-033401
TITLE: Preparing composition having lipid-encapsulated therapeutic agent particles, by combining lipid with buffered aqueous solution of charged therapeutic agent, changing pH of mixture to form lipid-encapsulated therapeutic agent particles.
DERWENT CLASS: A23 A25 A96 B04 D16
INVENTOR(S): ANSELL, S M; CULLIS, P; DEBEYER, D; HARASYM, T; HOPE, M J; KLIMUK, S K; SCHERRER, P; SEMPLE, S C
PATENT ASSIGNEE(S): (INEX-N) INEX PHARM CORP
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2005008689	A1	20050113	(200511)*		49

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 2005008689	A1	CIP of	US 1997-856374	19970514
		Cont of	US 1998-78954	19980514
		Cont of	US 2001-895480	20010629
			US 2004-925734	20040824

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 2005008689	A1	Cont of	US 6287591

PRIORITY APPLN. INFO: US 1998-78954 19980514; US 1997-856374 19970514; US 2001-895480 20010629; US 2004-925734 20040824
AN 2005-099932 [11] WPIDS
CR 1999-045179 [04]; 2001-226663 [23]; 2002-024658 [03]; 2003-874615 [81]; 2004-031296 [03]; 2005-072236 [08]; 2006-016984 [02]
AB US2005008689 A UPAB: 20060106
NOVELTY - Preparing (M1) composition comprising lipid-encapsulated therapeutic agent particles, by combining mixture of lipids having first lipid component and second lipid component with buffered aqueous solution of charged therapeutic agent to form intermediate mixture containing lipid-encapsulated therapeutic agent particles, and changing pH of mixture to provide partially-surface neutralized lipid-encapsulated therapeutic agent particles is new.

DETAILED DESCRIPTION - Preparing (M1) a composition comprising lipid-encapsulated therapeutic agent particles, by:

(a) combining a mixture of lipids having at least a first lipid component and a second lipid component with a buffered aqueous solution of a charged therapeutic agent to form an intermediate mixture containing lipid-encapsulated therapeutic agent particles, where the first lipid component is selected from among lipids containing a protonatable or deprotonatable group that has a pKa so that the lipid is in a charged form at a first pH and a neutral form

at a second pH, the buffered solution has a pH so that the first lipid component is in its charged form when in the buffered solution, the first lipid component is further selected so that the charged form is cationic when the charged therapeutic agent is anionic in the buffered solution, and anionic when the charged therapeutic agent is cationic in the buffered solution, and the second lipid component is selected from lipids that prevent particle aggregation during lipid-therapeutic agent particle formation; and

(b) changing the pH of the intermediate mixture to neutralize at least some exterior surface charges on the lipid-encapsulated therapeutic agent particles to provide at least partially-surface neutralized lipid-encapsulated therapeutic agent particles.

INDEPENDENT CLAIMS are also included for:

(1) a composition (I) comprising:

(a) lipid-therapeutic agent particles having a lipid portion and a charged therapeutic agent being encapsulated in the lipid **protein**, where the lipid portion comprises at least a first lipid component and a second lipid component, the first lipid component is selected from among lipids containing a protonatable or deprotonatable group that has a pKa so that the lipid is in a charged form at a first pH and a neutral form at a second pH, the first lipid component is further selected so that the charged form is cationic when the therapeutic agent is anionic and anionic when the therapeutic agent is cationic, and the second lipid component is selected from lipids that prevent particle aggregation during lipid-nucleic acid particle formation and which exchange out of the lipid particle at a rate greater than polyethylene glycol coupled to a ceramide derivative with 20 carbon acyl chain (PEG-CerC20);

(b) lipid-therapeutic agent particles having a lipid portion and a charged therapeutic agent being encapsulated in the lipid **protein** in (a), where the particles have a nucleic acid/lipid ratio (in %, by weight) (10) and a size of 70-200 nm; or

(c) a lipid-encapsulated nucleic acid particles, which contain nucleic acids (in %, by weight) (10), where the nucleic acids have exclusively phosphodiester linkages; and

(2) a composition (C1) comprising lipid-therapeutic agent particles prepared by (M1), and a carrier.

ACTIVITY - Cytostatic; Antiinflammatory; Antimicrobial.

In vivo analysis of the lipid-encapsulated c-myc antisense in reduction of tumor volume was carried out as follows. On day 0 of the study, 3 multiply 105 B 16/BL6 murine melanoma cells were injected subcutaneously into the dorsal flank of female C57BL/6 mice. Tumors were allowed to grow for a period of 5-7 days until the tumors reached 50-100 mm3 before initiating treatments with test sample or controls. Mice were dosed every other day for a total of 7 doses. Administrations were through intravenous tail vein injections. Test group was administered with AS4200 containing distearoylphosphatidylcholine, cholesterol, dioleoylphosphatidyldiaminopropane, polyethylene glycol-ceramide (PEG-CerC14) and c-myc antisense (LR-3280; AACGTTGAGGGGCAT), and the control group was administered with HBS buffer (N-(2-hydroxyethyl)piperazine-N'-ethanesulfonic acid (HEPES) (20 mM) and **sodium chloride** (145 mM)) or AS4200 not containing antisense. After treatment, at day 18, mice were euthanized and the tumor volume was noted. The test group containing AS4200 having c-myc antisense exhibited small ranges in tumor volumes (285-451 mm3), while the control group exhibited no inhibitory effect on tumor volumes.

MECHANISM OF ACTION - Reduces aberrant expression of intracellular adhesion molecule (ICAM)-1, c-myc, c-myb, ras, raf,

erb-B-2, **protein kinase** C (PKC)- alpha , insulin-like growth factor (IGF)-1R, epidermal growth factor receptor (EGFR), vascular endothelial growth factor (VEGF) and VEGF receptor 1 (VEGF-R-1) (claimed).

USE - (M1) is useful for preparing a composition comprising lipid-encapsulated therapeutic agent particles. (I) is useful for introducing a nucleic acid into a cell, by contacting a cell with (I), for a period of time sufficient to introduce the nucleic acid into the cell. (I) is useful for treating or preventing a disease characterized by aberrant expression of a gene in a mammalian subject, by preparing (I), where the therapeutic nucleic acid component **hybridizes** specifically with the aberrantly expressed gene, and administering the particle to the mammalian subject, where the expression of the aberrantly expressed gene is reduced, or administering (I) to mammalian subject, where (I) comprises lipid-encapsulated nucleic acid particles containing nucleic acids (in %, by weight) (10), where the nucleic acids have exclusively phosphodiester linkages. The gene is chosen from intracellular adhesion molecule (ICAM)-1, c-myc, c-myb, ras, raf, erb-B-2, **protein kinase** C (PKC)- alpha , insulin-like growth factor (IGF)-1R, epidermal growth factor receptor (EGFR), vascular endothelial growth factor (VEGF) and VEGF receptor 1 (VEGF-R-1). The disease is a tumor, inflammation or infectious disease. (I) is useful for preventing expression of a disease-associated gene in a mammalian cell, by preparing lipid-therapeutic oligonucleotide particle containing an antisense therapeutic agent, and exposing the mammalian cell to the lipid-therapeutic oligonucleotide particle for the therapeutic oligonucleotide component to enter the cell, where the antisense therapeutic agent has a sequence complementary to the disease-associated gene and reduces the production of the gene product of the disease-associated gene in the cell (all claimed).

ADVANTAGE - The therapeutic agent of (M1) is protected from degradation and clearance in serum.

DESCRIPTION OF DRAWING(S) - The figure is a graph representing results of a study on the in vivo efficacy of lipid-encapsulated antisense particles in a mouse tumor model.

Dwg.19/23

L22 ANSWER 6 OF 39 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:530582 BIOSIS
DOCUMENT NUMBER: PREV200510324097
TITLE: CART expression in the rat nucleus accumbens is regulated by the PKA/CREB pathway.
AUTHOR(S): Jones, Douglas Campbell [Reprint Author]; Kuhar, Michael J.
CORPORATE SOURCE: Emory Univ, Yerkes Natl Primate Res Ctr, Atlanta, GA 30329 USA
SOURCE: FASEB Journal, (MAR 4 2005) Vol. 19, No. 4, Suppl. S, Part 1, pp. A509.
Meeting Info.: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences. San Diego, CA, USA. March 31 -April 06, 2005. Amer Assoc Anatomists; Amer Assoc Immunologists; Amer Physiol Soc; Amer Soc Biochem & Mol Biol; Amer Soc Investigat Pathol; Amer Soc Nutr Sci; Amer Soc Pharmacol & Expt Therapeut; Int Union Physiol Sci.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005

AB Cocainc-amphetamine regulated transcript (CART) is involved in the brain's reward circuit. For instance, CART **peptides** regulate feeding behaviors and antagonize the behavioral effects of cocaine and amphetamines; consequently, the expression of CART must be strictly regulated. The CART gene has been extensivelycharacterized and binding sites for several transcription factors have been identified within the promoter region, including the cyclic AMP-response element (CRE), which serves as a binding site for cyclic-AMP responsive binding **protein** (CREB). CART expression appears to be regulated via the PKA/CREB pathway in cell cultures. Therefore, the goal of these studies was to examine the involvement of PKA/CREB in CART mRNA expression in-vivo in the rat nucleus accumbens. Intra-accumbal injections of forskolin, an adenylate cyclase and cAMP pathway activator, increased. CART mRNA expression measured by in-situ **hybridization**, whereas **saline** as well as an inactive forskolin analog had no effect on CART expression. Phosphorylation of CREB by **protein kinase** A (PKA) is a necessarystep for the activation of CREB. Inhibition of PKA with H89 and Rp-cAMPS, which have different mechanisms of action, attenuated forskolin-induced CART expression. These studies, therefore, are the first to provide evidence that CART expression in-vivo in the rat nucleus accumbens is regulated by the cAMP/PKA pathway, and ultimately through the activation of CREB.

L22 ANSWER 7 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2004-157117 [15] WPIDS
DOC. NO. CPI: C2004-062552
TITLE: New AKIP1 nucleic acids and proteins, useful in producing plants with improved response to stresses including cold, heat, salinity, synthetic and natural chemical agents, viral, fungal and bacterial pathogens and drought.
DERWENT CLASS: C06 D16
INVENTOR(S): ASSMANN, S M; KINOSHITA, T; MANSFIELD, J L; NG, C K Y; SHIMAZAKI, K
PATENT ASSIGNEE(S): (PENN-N) PENN STATE RES FOUND
COUNTRY COUNT: 104
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004013295	A2	20040212	(200415)*	EN	106
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2003265343	A1	20040223	(200453)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 2004013295	A2	WO 2003-US24197	20030801
AU 2003265343	A1	AU 2003-265343	20030801

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003265343	A1 Based on	WO 2004013295

PRIORITY APPLN. INFO: US 2002-400549P 20020801
AN 2004-157117 [15] WPIDS
AB WO2004013295 A UPAB: 20040302
NOVELTY - An isolated nucleic acid molecule (I) is new.

DETAILED DESCRIPTION - An isolated nucleic acid molecule (I) encodes a plant RNA binding protein, where the protein comprises an RNA recognition motif selected from a sequences of 82 or 81 amino acids (SEQ ID NOS: 22-24), and further comprises one or more sequences selected from any of the 6 sequences of 20-86 amino acids (SEQ ID NOS: 25-30), where the binding of RNA by the encoded RNA binding protein is abscisic acid (ABA)-mediated and where accumulation of a transcript for an mRNA encoding the plant RNA binding protein is not mediated by ABA, where the encoded RNA binding protein further comprises: Xaa-Phe-Xaa-Xaa-Gly-Leu; Ile-Phe-Val-His-Gly-Leu; Lys-Gly-Tyr-Xaa-Phe-Xaa-Xaa-Xaa; or Lys-Gly-Tyr-Gly-Phe-Ile-Leu-Xaa (SEQ ID NOS: 31-34).

INDEPENDENT CLAIMS are also included for:

(1) a vector comprising (I);
(2) a plant cell comprising the vector of (1);
(3) a transgenic plant comprising the vector of (1);
(4) a protein produced by the expression of (I);
(5) an antibody specific for the protein of (4);
(6) an oligonucleotide having at least 15 consecutive nucleotides identical in sequence to a consecutive 15 nucleotide sequence of (I);
(7) a genetically altered plant, having altered response to ABA as compared with an unaltered control plant, comprising an ABA-mediated phosphorylation-regulated RNA binding protein that is substantially nonfunctional or absent or that is increased in amount or activity as compared with the control plant;
(8) a method for improving a plant's ABA-regulated response to a stressor;
(9) a method to alter the expression or activity of a stress-related protein in a plant;
(10) a method to alter ABA sensitivity in a plant;
(11) a fertile plant produced by the method of (10); and
(12) an isolated plant protein having RNA binding properties, where the protein is of a length of 400-525 amino acids and where the RNA binding properties are regulated by phosphorylation of the protein.

ACTIVITY - Plant Protectant. No suitable biological data provided.

MECHANISM OF ACTION - None Given.

USE - The nucleic acid molecule and the encoded protein is useful in producing plants with improved response to stresses including cold, heat, salinity, synthetic and natural chemical agents, viral, fungal and bacterial pathogens and drought.

Dwg.0/8

L22 ANSWER 8 OF 39 MEDLINE on STN
ACCESSION NUMBER: 2004491753 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15460057

TITLE: Expression of nerve growth factor mRNA and nerve regeneration after discontinuous injection of phorbol-12-myristate-13-acetate into silicone chamber.
AUTHOR: Jin Y; Luo Yongxiang; Zheng Jia
CORPORATE SOURCE: Department of Orthopedic Surgery, Henan Provincial Hospital, Zhengzhou Henan 450003, PR China.
SOURCE: Zhongguo xiu fu chong jian wai ke za zhi = Zhongguo xiufu chongjian waike zazhi = Chinese journal of reparative and reconstructive surgery, (2004 Sep) Vol. 18, No. 5, pp. 409-13.
Journal code: 9425194. ISSN: 1002-1892.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200503
ENTRY DATE: Entered STN: 20041006
Last Updated on STN: 20050326
Entered Medline: 20050325

AB OBJECTIVE: To study the mRNA expressions of **protein kinase** C(PKC) and nerve growth factor (NGF) in rat sciatic nerve and the number of axons after phorbol-12-myristate-13-acetate (PMA) was injected into silicone chamber. METHODS: Forty-two SD adult rats were divided into six groups depending on the time of injury (1 day, 3 days, 1 week, 2 weeks, 3 weeks and 4 weeks). A 0.5 cm nerve was cut in double rat sciatic nerves and "T" type silicone chamber was sutured. PMA at the concentration of 1 x 10(-9) mol/L was injected discontinuously into the right side of T type silicone chamber (PMA group) and **saline** was injected into the left side (control group). Nucleic acid in situ **hybridization** histochemistry technique and the computer imagine analysis were employed to detect dynamic changes of PKC mRNA and NGF mRNA in rat sciatic nerves. The number of axons was measured. RESULTS: The expressions of PKC mRNA and NGF mRNA increased after injury, and the expressions of PKC mRNA and NGF mRNA reached the peak 2 weeks and 3 weeks after injury respectively in control group. The expressions of PKC mRNA and NGF mRNA in PMA group were significantly increased than those in control group 2,3 and 4 weeks after injury ($P < 0.01$). The number of axons in PMA group significantly increased than that in control group ($P < 0.01$). CONCLUSION: PKC involved in the expression of NGF mRNA and nerve regeneration after injury. During the regenerated course, PMA can promote the expression of NGF mRNA and the number of axons after injury.

L22 ANSWER 9 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-748334 [70] WPIDS
DOC. NO. CPI: C2003-205210
TITLE: New pharmaceutical composition comprising a nucleic acid molecule encoding proteins regulating the energy homeostasis and metabolism of triglycerides useful for detecting or preventing metabolic diseases, e.g. obesity.
DERWENT CLASS: B04 D16
INVENTOR(S): BRONNER, G; EULENBERG, K; HADER, T; STEUERNAGEL, A; BROENNER, G; HAEDER, T
PATENT ASSIGNEE(S): (DEVE-N) DEVELOGEN ENTWICKLUNGSBIOLOGISCHE FORSCH; (DEVE-N) DEVELOGEN ENTWICKLUNGSBIOLOGISC AG
COUNTRY COUNT: 104
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003075945	A2	20030918	(200370)*	EN	140
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2003227071	A1	20030922	(200431)		
EP 1492553	A2	20050105	(200504)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
US 2005119206	A1	20050602	(200537)		
JP 2005519947	W	20050707	(200545)		75

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003075945	A2	WO 2003-EP2714	20030314
AU 2003227071	A1	AU 2003-227071	20030314
EP 1492553	A2	EP 2003-743885	20030314
		WO 2003-EP2714	20030314
US 2005119206	A1	WO 2003-EP2714	20030314
		US 2004-507617	20040914
JP 2005519947	W	JP 2003-574218	20030314
		WO 2003-EP2714	20030314

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2003227071	A1	Based on	WO 2003075945
EP 1492553	A2	Based on	WO 2003075945
JP 2005519947	W	Based on	WO 2003075945

PRIORITY APPLN. INFO: EP 2002-6810 20020325; EP 2002-5882 20020314; EP 2002-6012 20020315; EP 2002-6271 20020320

AN 2003-748334 [70] WPIDS

AB WO2003075945 A UPAB: 20031030

NOVELTY - A pharmaceutical composition comprising:

(a) a nucleic acid molecule (I) of the SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 gene family or its encoded polypeptide (II) or a fragment or variant of the nucleic acid molecule or polypeptide;

(b) an effector, e.g. an antibody;

(c) an aptamer; or

(d) another receptor recognizing (I) or (II), is new.

DETAILED DESCRIPTION - A pharmaceutical composition comprising:

(a) a nucleic acid molecule (I) of the SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 gene family or its encoded polypeptide (II) or a fragment or variant of the nucleic acid molecule or polypeptide;

(b) an effector, e.g. an antibody;

(c) an aptamer; or

(d) another receptor recognizing the nucleic acid molecule or its encoded polypeptide, preferably together with carriers, diluents and/or adjuvants, is new.

INDEPENDENT CLAIMS are also included for:

(1) a non-human transgenic animal exhibiting a modified expression of a SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 homologous polypeptide;

(2) a recombinant host cell exhibiting a modified expression of a SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, FtsJ2, AWP1, or ZNF216 homologous polypeptide;

(3) identifying a (poly)peptide involved in the regulation of energy homeostasis and/or metabolism of triglycerides in a mammal;

(4) screening for an agent which modulates the interaction of a SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, FtsJ2, AWP1, or ZNF216 homologous polypeptide with a binding target/agent;

(5) screening for an agent which modulates the activity of SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, FtsJ2, AWP1 or ZNF216 homologous polypeptide;

(6) producing a composition comprising the (poly)peptide identified by method of (3) or agent identified by method of (4) or (5) with a carrier, diluent or adjuvant; and

(7) a kit comprising:

(a) a SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, FtsJ2, AWP1, or ZNF216 nucleic acid molecule or its fragment;

(b) a vector comprising the nucleic acid of (a);

(c) a host cell comprising the nucleic acid of (a) or the vector of (b);

(d) a polypeptide encoded by the nucleic acid of (1);

(e) a fusion polypeptide encoded by the nucleic acid of (a);

(f) an antibody, an aptamer or another receptor against the nucleic acid of (a) or the polypeptide of (d) or (e); and

(g) an anti-sense oligonucleotide of the nucleic acid of (a).

ACTIVITY - Anorectic; Immunomodulator; Antidiabetic; Hypotensive; Cardiant; Osteopathic; Antilipemic. No biological data given.

MECHANISM OF ACTION - Gene Therapy. No biological data given.

USE - The composition is useful for the manufacture of an agent for detecting, verifying, treating, alleviating or preventing disorders, including metabolic diseases such as obesity and other body-weight regulation disorders as well as related disorders such as metabolic syndrome, eating disorder, cachexia, diabetes mellitus, hypertension, coronary heart disease, hypercholesterolemia, dyslipidemia, osteoarthritis or gallstones, in cells, cell masses, organs or subjects. A nucleic acid molecule (I) of the SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 gene family or a fragment is used for the preparation of a non-human animal which over or under expresses the SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 gene product. (I) of the SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 gene family or its encoded polypeptide (II) or a fragment or variant of the nucleic acid molecule or polypeptide; an effector, e.g. an antibody; an aptamer; or another receptor recognizing (I) or (II), is used for identifying substances that interact with a SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 homologous polypeptide. (I) of the SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 gene family or its encoded polypeptide (II) or a fragment or variant of the nucleic acid

molecule or polypeptide; an effector, e.g. an antibody; an aptamer; or another receptor recognizing (I) or (II) is used for controlling the function of a gene and/or gene product which is influenced and/or modified by a SRM, NPFF2, NPFF1, OX1R, OX2R, SLC25A11, PRKAR2A, PRKAR2B, Cab45, Fts J2, AWP1, or ZNF216 homologous polypeptide (all claimed).
Dwg.0/27

L22 ANSWER 10 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-679632 [64] WPIDS
CROSS REFERENCE: 2003-092892 [08]; 2003-671659 [63]; 2003-875735 [81]
DOC. NO. CPI: C2003-185723
TITLE: New nucleic acid molecule, useful for preparing a composition for treating cancer.
DERWENT CLASS: B04 D16
INVENTOR(S): ALFANO, J R; CARTINHOUR, S W; COLLMER, A; SCHNEIDER, D J; TANG, X
PATENT ASSIGNEE(S): (CORR) CORNELL RES FOUND INC; (UNIV) UNIV KANSAS STATE RES FOUND; (UYNE-N) UNIV NEBRASKA; (USDA) US SEC OF AGRIC
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003068930	A2	20030821	(200364)*	EN	180
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW					
AU 2003216273	A1	20030904	(200428)		
AU 2003216273	A8	20051020	(200615)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003068930	A2	WO 2003-US4450	20030212
AU 2003216273	A1	AU 2003-216273	20030212
AU 2003216273	A8	AU 2003-216273	20030212

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003216273	A1 Based on	WO 2003068930
AU 2003216273	A8 Based on	WO 2003068930

PRIORITY APPLN. INFO: US 2002-380185P 20020510; US 2002-356408P 20020212
AN 2003-679632 [64] WPIDS
CR 2003-092892 [08]; 2003-671659 [63]; 2003-875735 [81]
AB WO2003068930 A UPAB: 20060302
NOVELTY - An isolated nucleic acid molecule comprising:
(a) a sequence encoding a polypeptide having a sequence comprising 130-1957 amino acids; or

(b) a sequence that hybridizes with a DNA molecule complementary to a sequence comprising 366-5874 bp under stringency conditions comprising a hybridization medium that includes 0.9 multiply **saline sodium citrate** (SSC) at a temperature of 42 deg. C, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an expression system comprising a vector into which is inserted the DNA molecule;

(2) a host cell comprising the DNA molecule;

(3) a transgenic plant comprising the DNA molecule;

(4) a composition comprising a carrier and the polypeptide;

(5) making a transgenic plant cell;

(6) imparting disease resistance to a plant;

(7) making a plant hypersusceptible to colonization by nonpathogenic bacteria;

(8) causing eukaryotic cell death;

(9) inhibiting programmed cell death;

(10) treating a cancerous condition; and

(11) modifying a metabolic pathway in a cell.

ACTIVITY - Cytostatic. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The nucleic acid molecule is useful for preparing a composition for treating cancer (claimed).

Dwg.0/7

L22 ANSWER 11 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-851360 [79] WPIDS
CROSS REFERENCE: 1995-382985 [49]; 1997-503105 [46]; 1998-286866 [25]; 1999-095340 [08]; 1999-229499 [19]; 1999-229532 [19]; 1999-229533 [19]; 1999-244420 [20]; 1999-254218 [21]; 1999-254381 [21]; 1999-254713 [21]; 1999-263480 [22]; 1999-302739 [25]; 1999-326705 [27]; 1999-337420 [28]; 1999-347718 [29]; 1999-371118 [31]; 1999-404743 [34]; 1999-430385 [36]; 1999-551358 [46]; 1999-580306 [49]; 1999-620728 [53]; 2000-038358 [03]; 2000-062031 [05]; 2000-072883 [06]; 2000-116314 [10]; 2000-237871 [20]; 2000-271386 [23]; 2000-271431 [23]; 2000-271434 [23]; 2000-271435 [23]; 2000-292842 [25]; 2000-317943 [27]; 2000-412154 [35]; 2000-412324 [35]; 2000-412325 [35]; 2000-430397 [37]; 2000-431586 [37]; 2000-442668 [38]; 2000-452188 [39]; 2000-452395 [39]; 2000-499263 [44]; 2000-565502 [52]; 2000-572269 [53]; 2000-572270 [53]; 2000-572271 [53]; 2000-587437 [55]; 2000-594320 [56]; 2000-594321 [56]; 2000-611443 [58]; 2000-611444 [58]; 2000-628263 [60]; 2000-638138 [61]; 2000-638201 [61]; 2000-679484 [66]; 2001-016509 [02]; 2001-025022 [03]; 2001-025251 [03]; 2001-025253 [03]; 2001-032160 [04]; 2001-050025 [06]; 2001-050091 [06]; 2001-070561 [08]; 2001-071075 [08]; 2001-071078 [08]; 2001-071395 [08]; 2001-081051 [09]; 2001-090793 [10]; 2001-091968 [10]; 2001-103149 [11]; 2001-183260 [18]; 2001-226690 [23]; 2001-226823 [23]; 2001-235264 [24]; 2001-381383 [40]; 2001-381384 [40]; 2001-408281 [43]; 2001-451708 [48]; 2001-541567 [60]; 2001-541628 [60]; 2001-602746 [68]; 2001-611580 [70]; 2001-625876 [72]; 2002-075461 [10]; 2002-090516 [12]; 2002-130120 [17]; 2002-130151 [17]; 2002-130882 [17]; 2002-171999 [22]; 2002-172001 [22]; 2002-205567 [26]; 2002-256031 [30]; 2002-266521 [31];

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Searcher : Shears 571-272-2528

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Searcher : Shears 571-272-2528

2003-540172 [51]; 2003-540605 [51]; 2003-540606 [51]; 2003-540607 [51]; 2003-540608 [51]; 2003-540609 [51]; 2003-540610 [51]; 2003-540611 [51]; 2003-540612 [51]; 2003-540613 [51]; 2003-540670 [51]; 2003-540671 [51]; 2003-540672 [51]; 2003-540675 [51]; 2003-540676 [51]; 2003-540677 [51]; 2003-540678 [51]; 2003-540681 [51]; 2003-540682 [51]; 2003-540684 [51]; 2003-540693 [51]; 2003-540697 [51]; 2003-555149 [52]; 2003-555150 [52]; 2003-555151 [52]; 2003-555152 [52]; 2003-555153 [52]; 2003-555154 [52]; 2003-555155 [52]; 2003-555156 [52]; 2003-555157 [52]; 2003-555158 [52]; 2003-555159 [52]; 2003-555475 [52]; 2003-555478 [52]; 2003-555481 [52]; 2003-555482 [52]; 2003-555483 [52]; 2003-555484 [52]; 2003-555580 [52]; 2003-555594 [52]; 2003-555595 [52]; 2003-555602 [52]; 2003-559089 [52]; 2003-567064 [53]; 2003-567065 [53]; 2003-567066 [53]; 2003-567176 [53]; 2003-567181 [53]; 2003-567182 [53]; 2003-567183 [53]; 2003-567184 [53]; 2003-567185 [53]; ...

DOC. NO. CPI: C2003-239767

TITLE: New isolated nucleic encoding guanylate binding protein-4, useful as hybridization probes, in chromosome and gene mapping, treating cancer, e.g. gastric cancer or melanoma or combating immunological and inflammatory responses.

DERWENT CLASS: B04 D16

INVENTOR(S): PENNICA, D

PATENT ASSIGNEE(S): (GETH) GENENTECH INC

COUNTRY COUNT: 1

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6642024	B1	20031104	(200379)*		109

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 6642024	B1 Cont of	US 1998-15089	19980129
		US 2000-643657	20000817

PRIORITY APPLN. INFO: US 1998-15089 19980129; US 2000-643657 20000817

AN 2003-851360 [79] WPIDS

CR 1995-382985 [49]; 1997-503105 [46]; 1998-286866 [25]; 1999-095340 [08]; 1999-229499 [19]; 1999-229532 [19]; 1999-229533 [19]; 1999-244420 [20]; 1999-254218 [21]; 1999-254381 [21]; 1999-254713 [21]; 1999-263480 [22]; 1999-302739 [25]; 1999-326705 [27]; 1999-337420 [28]; 1999-347718 [29]; 1999-371118 [31]; 1999-404743 [34]; 1999-430385 [36]; 1999-551358 [46]; 1999-580306 [49]; 1999-620728 [53]; 2000-038358 [03]; 2000-062031 [05]; 2000-072883 [06]; 2000-116314 [10]; 2000-237871 [20]; 2000-271386 [23]; 2000-271431 [23]; 2000-271434 [23]; 2000-271435 [23]; 2000-292842 [25]; 2000-317943 [27]; 2000-412154 [35]; 2000-412324 [35]; 2000-412325 [35]; 2000-430397 [37]; 2000-431586 [37]; 2000-442668 [38]; 2000-452188 [39]; 2000-452395 [39]; 2000-499263 [44]; 2000-565502 [52]; 2000-572269 [53]; 2000-572270 [53]; 2000-572271 [53]; 2000-587437 [55]; 2000-594320 [56]; 2000-594321 [56];

2000-611443 [58]; 2000-611444 [58]; 2000-628263 [60]; 2000-638138 [61]; 2000-638201 [61]; 2000-679484 [66]; 2001-016509 [02]; 2001-025022 [03]; 2001-025251 [03]; 2001-025253 [03]; 2001-032160 [04]; 2001-050025 [06]; 2001-050091 [06]; 2001-070561 [08]; 2001-071075 [08]; 2001-071078 [08]; 2001-071395 [08]; 2001-081051 [09]; 2001-090793 [10]; 2001-091968 [10]; 2001-103149 [11]; 2001-183260 [18]; 2001-226690 [23]; 2001-226823 [23]; 2001-235264 [24]; 2001-381383 [40]; 2001-381384 [40]; 2001-408281 [43]; 2001-451708 [48]; 2001-541567 [60]; 2001-541628 [60]; 2001-602746 [68]; 2001-611580 [70]; 2001-625876 [72]; 2002-075461 [10]; 2002-090516 [12]; 2002-130120 [17]; 2002-130151 [17]; 2002-130882 [17]; 2002-171999 [22]; 2002-172001 [22]; 2002-205567 [26]; 2002-256031 [30]; 2002-266521 [31]; 2002-280917 [32]; 2002-280928 [32]; 2002-280940 [32]; 2002-292065 [33]; 2002-362426 [39]; 2002-383270 [41]; 2002-392224 [42]; 2002-404358 [43]; 2002-487624 [52]; 2002-641562 [69]; 2002-657277 [70]; 2002-665999 [71]; 2002-673016 [72]; 2002-673823 [72]; 2002-690475 [74]; 2002-697823 [75]; 2002-713224 [77]; 2002-731348 [79]; 2002-740172 [80]; 2002-750461 [81]; 2003-018887 [01]; 2003-066231 [06]; 2003-066810 [06]; 2003-066893 [06]; 2003-066898 [06]; 2003-090845 [08]; 2003-101755 [09]; 2003-102117 [09]; 2003-147110 [14]; 2003-147434 [14]; 2003-147446 [14]; 2003-148238 [14]; 2003-155950 [15]; 2003-167072 [16]; 2003-167406 [16]; 2003-174088 [17]; 2003-174140 [17]; 2003-174141 [17]; 2003-174176 [17]; 2003-174358 [17]; 2003-175282 [17]; 2003-183819 [18]; 2003-183820 [18]; 2003-183821 [18]; 2003-183822 [18]; 2003-198285 [19]; 2003-201194 [19]; 2003-238305 [23]; 2003-247083 [24]; 2003-275292 [27]; 2003-275322 [27]; 2003-288105 [28]; 2003-288106 [28]; 2003-288123 [28]; 2003-288142 [28]; 2003-288163 [28]; 2003-311003 [30]; 2003-328338 [31]; 2003-328481 [31]; 2003-328482 [31]; 2003-328499 [31]; 2003-328592 [04]; 2003-328610 [31]; 2003-328612 [04]; 2003-328636 [31]; 2003-328637 [31]; 2003-328851 [06]; 2003-328857 [31]; 2003-328860 [31]; 2003-328863 [31]; 2003-329601 [31]; 2003-329602 [31]; 2003-330485 [31]; 2003-330984 [31]; 2003-331419 [31]; 2003-331420 [31]; 2003-331421 [31]; 2003-331422 [31]; 2003-331484 [31]; 2003-331485 [31]; 2003-331911 [31]; 2003-331912 [31]; 2003-331913 [31]; 2003-331914 [31]; 2003-331915 [31]; 2003-331916 [31]; 2003-331917 [31]; 2003-331918 [31]; 2003-331919 [31]; 2003-331920 [31]; 2003-331921 [31]; 2003-331922 [31]; 2003-331923 [31]; 2003-331924 [31]; 2003-331925 [31]; 2003-332024 [31]; 2003-332025 [31]; 2003-332026 [31]; 2003-332027 [31]; 2003-332028 [31]; 2003-332029 [31]; 2003-332030 [31]; 2003-332031 [31]; 2003-332032 [31]; 2003-332033 [31]; 2003-332034 [31]; 2003-332035 [31]; 2003-332036 [31]; 2003-332037 [31]; 2003-332038 [31]; 2003-332039 [31]; 2003-332040 [31]; 2003-332052 [31]; 2003-340824 [32]; 2003-340981 [32]; 2003-341079 [32]; 2003-341189 [32]; 2003-341326 [32]; 2003-341327 [32]; 2003-341328 [32]; 2003-341350 [32]; 2003-341502 [32]; 2003-341586 [32]; 2003-341587 [32]; 2003-341588 [14]; 2003-341589 [32]; 2003-341590 [32]; 2003-341591 [32]; 2003-341713 [32]; 2003-341714 [32]; 2003-341840 [32]; 2003-341960 [32]; 2003-341961 [32]; 2003-341962 [32]; 2003-341963 [32]; 2003-341964 [32]; 2003-341965 [32]; 2003-341966 [32]; 2003-341967 [32]; 2003-341968 [32]; 2003-341969 [32]; 2003-341970 [32]; 2003-341971 [32]; 2003-341972 [32]; 2003-341973 [32]; 2003-341974 [32]; 2003-341975 [32]; 2003-341976 [32]; 2003-341977 [32]; 2003-341978 [32]; 2003-341979 [32]; 2003-341980 [32]; 2003-342036 [32]; 2003-342037 [32]; 2003-342038 [32]; 2003-342039 [32]; 2003-342040 [32]; 2003-342041 [32]; 2003-342042 [32]; 2003-342045 [32]; 2003-342069 [32]; 2003-342070 [32]; 2003-344635

[33]; 2003-352829 [33]; 2003-352836 [33]; 2003-353471 [33]; 2003-353472 [33]; 2003-353754 [33]; 2003-361832 [34]; 2003-362213 [34]; 2003-362743 [34]; 2003-362802 [34]; 2003-362803 [34]; 2003-362804 [34]; 2003-370792 [35]; 2003-370793 [35]; 2003-370797 [35]; 2003-392892 [37]; 2003-393229 [37]; 2003-401699 [38]; 2003-401702 [38]; 2003-401847 [38]; 2003-401848 [38]; 2003-401849 [38]; 2003-401974 [38]; 2003-402071 [38]; 2003-402072 [38]; 2003-402073 [38]; 2003-402074 [38]; 2003-402075 [38]; 2003-402076 [38]; 2003-402077 [38]; 2003-402282 [38]; 2003-402283 [38]; 2003-417249 [39]; 2003-417250 [39]; 2003-417251 [39]; 2003-417252 [39]; 2003-417284 [39]; 2003-417923 [39]; 2003-417924 [39]; 2003-418401 [39]; 2003-428843 [40]; 2003-439036 [41]; 2003-439037 [41]; 2003-439038 [41]; 2003-447383 [42]; 2003-447384 [42]; 2003-447409 [42]; 2003-447471 [42]; 2003-447472 [42]; 2003-456261 [43]; 2003-456352 [43]; 2003-456353 [43]; 2003-456354 [43]; 2003-456358 [43]; 2003-456484 [43]; 2003-456485 [43]; 2003-456486 [43]; 2003-466223 [44]; 2003-466224 [44]; 2003-466225 [44]; 2003-466226 [44]; 2003-466308 [44]; 2003-466344 [44]; 2003-466345 [44]; 2003-466346 [44]; 2003-466347 [44]; 2003-466348 [44]; 2003-466349 [44]; 2003-466350 [44]; 2003-466351 [44]; 2003-466352 [44]; 2003-466353 [44]; 2003-466354 [44]; 2003-466355 [44]; 2003-466405 [44]; 2003-466444 [44]; 2003-466445 [44]; 2003-466446 [44]; 2003-466447 [44]; 2003-466448 [44]; 2003-466449 [44]; 2003-479475 [45]; 2003-479537 [45]; 2003-479538 [45]; 2003-479539 [45]; 2003-479540 [45]; 2003-479637 [45]; 2003-479638 [45]; 2003-479639 [45]; 2003-479640 [45]; 2003-479641 [45]; 2003-479642 [45]; 2003-479681 [45]; 2003-479682 [45]; 2003-479683 [45]; 2003-479684 [45]; 2003-479685 [45]; 2003-479686 [45]; 2003-479687 [45]; 2003-479724 [45]; 2003-479740 [45]; 2003-479741 [45]; 2003-479742 [45]; 2003-479743 [45]; 2003-479873 [45]; 2003-479874 [45]; 2003-479875 [45]; 2003-479876 [45]; 2003-492030 [46]; 2003-492032 [46]; 2003-492150 [46]; 2003-492169 [46]; 2003-492170 [46]; 2003-492256 [46]; 2003-492257 [46]; 2003-492258 [46]; 2003-492259 [46]; 2003-492260 [46]; 2003-492261 [46]; 2003-492263 [46]; 2003-492264 [46]; 2003-492265 [46]; 2003-492266 [46]; 2003-492267 [46]; 2003-492268 [46]; 2003-492269 [46]; 2003-492270 [46]; 2003-492271 [46]; 2003-492272 [46]; 2003-492273 [46]; 2003-492274 [46]; 2003-492275 [46]; 2003-492350 [46]; 2003-503296 [47]; 2003-503297 [47]; 2003-503301 [47]; 2003-503391 [47]; 2003-503392 [47]; 2003-503393 [47]; 2003-503396 [47]; 2003-503399 [47]; 2003-503400 [47]; 2003-503401 [47]; 2003-503402 [47]; 2003-503574 [47]; 2003-503575 [47]; 2003-503626 [47]; 2003-503627 [47]; 2003-503628 [47]; 2003-503629 [47]; 2003-503630 [47]; 2003-503631 [47]; 2003-503632 [47]; 2003-503633 [47]; 2003-512315 [48]; 2003-512316 [48]; 2003-521626 [49]; 2003-521627 [49]; 2003-521628 [49]; 2003-521801 [49]; 2003-521802 [49]; 2003-521803 [49]; 2003-521804 [49]; 2003-521814 [49]; 2003-521818 [49]; 2003-521819 [49]; 2003-521820 [49]; 2003-521821 [49]; 2003-521848 [49]; 2003-521849 [49]; 2003-521850 [49]; 2003-521851 [49]; 2003-521852 [49]; 2003-521853 [49]; 2003-521854 [49]; 2003-521859 [49]; 2003-521860 [49]; 2003-521865 [49]; 2003-521866 [49]; 2003-521911 [49]; 2003-521927 [49]; 2003-522018 [49]; 2003-531419 [50]; 2003-531434 [50]; 2003-531471 [50]; 2003-531472 [50]; 2003-531657 [50]; 2003-531719 [50]; 2003-531720 [50]; 2003-531721 [50]; 2003-531722 [50]; 2003-531737 [50]; 2003-540172 [51]; 2003-540605 [51]; 2003-540606 [51]; 2003-540607 [51]; 2003-540608 [51]; 2003-540609 [51]; 2003-540610 [51]; 2003-540611 [51]; 2003-540612 [51]; 2003-540613 [51]; 2003-540670 [51]; 2003-540671 [51]; 2003-540672 [51]; 2003-540675 [51]; 2003-540676 [51];

2003-540677 [51]; 2003-540678 [51]; 2003-540681 [51]; 2003-540682 [51]; 2003-540684 [51]; 2003-540693 [51]; 2003-540697 [51]; 2003-555149 [52]; 2003-555150 [52]; 2003-555151 [52]; 2003-555152 [52]; 2003-555153 [52]; 2003-555154 [52]; 2003-555155 [52]; 2003-555156 [52]; 2003-555157 [52]; 2003-555158 [52]; 2003-555159 [52]; 2003-555475 [52]; 2003-555478 [52]; 2003-555481 [52]; 2003-555482 [52]; 2003-555483 [52]; 2003-555484 [52]; 2003-555580 [52]; 2003-555594 [52]; 2003-555595 [52]; 2003-555602 [52]; 2003-559089 [52]; 2003-567064 [53]; 2003-567065 [53]; 2003-567066 [53]; 2003-567176 [53]; 2003-567181 [53]; 2003-567182 [53]; 2003-567183 [53]; 2003-567184 [53]; 2003-567185 [53]; ...

AB US 6642024 B UPAB: 20060206

NOVELTY - An isolated nucleic acid (I) comprising DNA which:

(a) encodes a guanylate binding **protein**-4 (GBP-4) **polypeptide** (II) comprising a sequence of 591 amino acids (designated as P1);

(b) has at least 95% identity to a DNA encoding (II);

(c) is a cDNA in ATCC deposit number 209456;

(d) **hybridizes** under stringent conditions with DNA encoding (II); or

(e) encodes (II) that binds to at least one guanine nucleotide, is new.

DETAILED DESCRIPTION - (I) may also comprise a DNA which encodes (II) comprising:

(a) a Asp-Thr-Glu-Gly (amino acid residues 97-100 of P1) GTP-binding consensus motif;

(b) a Thr-Leu-Arg-Asp (amino acid residues 179-182 of P1) potential casein kinase II phosphorylation site;

(c) a Ser-Gly-Lys-Glu (amino acid residues 568-571 of P1) potential casein kinase II phosphorylation site;

(d) a Thr-Leu-Arg (amino acid residues 179-181 of P1) potential **protein kinase** C phosphorylation site;

(e) a Thr-Met-Arg (amino acid residues 562-564 of P1) potential **protein kinase** C phosphorylation site;

(f) a Ser-Gly-Lys (amino acid residues 568-570 of P1) potential **protein kinase** C phosphorylation site;

(g) a Ser-Gln-Lys (amino acid residues 586-588 of P1) potential **protein kinase** C phosphorylation site;

(h) a Gly-Ile-Met-Val-Asn-Gly (amino acid residues 283-288 of P1) potential **protein kinase** C phosphorylation site;

(i) a Gly-Ser-Gln-Gln-Gly-Val (amino acid residues 579-584 of P1) potential N-myristoylation site; or

(j) a Cys-Phe-Ile-Ser (amino acid residues 554-557 of P1) potential prenylation site, where the stringent condition are 0.015 M **sodium chloride**/0.0015 M **sodium citrate**/0.1% sodium dodecyl sulfate at 50 deg. C.

INDEPENDENT CLAIMS are also included for the following:

(1) a vector comprising (I);

(2) a host cell comprising the vector of (1); and

(3) a process of producing a GBP-4 **polypeptide** comprising culturing the host cell of (2) under conditions suitable for expression of the GBP-4 **polypeptide** and recovering the GBP-4 **polypeptide** from the cell culture.

ACTIVITY - Neuroprotective; Cytostatic.

No biological data given.

MECHANISM OF ACTION - Immunostimulant.

USE - The nucleic acid and the encoded GBP-4 **polypeptide**, the antibodies and the composition are useful in treating myelodysplastic disorders, myeloproliferative syndromes, acute myeloid

leukemia and cancer, e.g. gastric, lung or colon cancer or melanoma. The nucleic acids and the encoded GBP-4 **polypeptide** are useful as **hybridization** probes, in chromosome and gene mapping, in generating transgenic animals, in radioimmunoassays, in inducing formation of anti-GBP-4 antibodies, in combating immunological and inflammatory responses and other pathological conditions like multiple sclerosis and lung and intestinal-related disorders, as mediator of any interferon-gamma-induced responses in macrophages and fibroblasts and may also function in other immune cell populations or in **protein** processing.
Dwg.0/8

L22 ANSWER 12 OF 39 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003408007 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12946798
TITLE: cDNA profiling in leukocytes exposed to hypertonic resuscitation fluids.
AUTHOR: Gushchin Vadim; Alam Hasan B; Rhee Peter; Kirkpatrick John R; Koustova Elena
CORPORATE SOURCE: Department of Surgery, Washington Hospital Center, Washington, DC, USA.
SOURCE: Journal of the American College of Surgeons, (2003 Sep) Vol. 197, No. 3, pp. 426-32.
Journal code: 9431305. ISSN: 1072-7515.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200309
ENTRY DATE: Entered STN: 20030830
Last Updated on STN: 20031001
Entered Medline: 20030930

AB BACKGROUND: Resuscitative fluids induce distinctive changes in leukocyte functions: incubation with colloid Dextran increases production of reactive oxygen species and adhesion, whereas exposure to hypertonic **saline** (HTS) inhibits "oxidative burst" and phagocytosis. In hypertonic **saline** Dextran (HTD), the hypertonic component determines the leukocyte functional behavior and subsequently activation response. We investigated whether leukocyte gene expression is analogously affected. METHODS: Whole blood from eight volunteers was diluted and incubated for 30 min at 37(o)C in 6.0% Dextran-70, 7.5% HTS, and 7.5% HTD. Total leukocyte RNA was extracted and used to synthesize biotinylated cDNA probes. Each probe was individually **hybridized** to a cDNA array to simultaneously measure the expression of 23 genes involved in inflammation, cell migration, and apoptosis. RESULTS: Leukocytes incubated with Dextran-70 demonstrated greater than a 6-fold ($p < 0.05$) increase in the expression of interleukin-8, growth-regulated oncogenes alpha and beta, L-selectin, superoxide dismutase, tumor necrosis factor-alpha (TNF-alpha), and mitogen-activated **protein kinase** 3. The expression profile induced by HTS was not significantly different from that of unstimulated blood, except for prominent induction of only three genes. HTD attenuated the expression of Dextran-70 upregulated genes, although the level of their expression was higher than in HTS-treated leukocytes. CONCLUSIONS: Hypertonic resuscitation fluids diminish the expression of immune activation-associated genes. Hypertonic component of HTD determines the leukocyte gene expression profile.

L22 ANSWER 13 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-723266 [78] WPIDS
DOC. NO. CPI: C2002-204755
TITLE: New isolated polynucleotide encoding at least one regulator of morphology capable of regulating the morphology of a dimorphic fungal cell, useful for producing and/or secreting large quantities of commercially valuable proteins.
DERWENT CLASS: B04 D16
INVENTOR(S): APPEL, K F; ARNAU, J; JACOBSEN, M D; PETERSEN, J B; POULSEN, U; WOLFF, A M; AMAU, J
PATENT ASSIGNEE(S): (BIOT-N) BIOTEKNOLOGISK INST; (ARNA-I) ARNAU J; (AMAU-I) AMAU J
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002070721	A2	20020912	(200278)*	EN	295
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW					
US 2003134353	A1	20030717	(200348)		
EP 1379666	A2	20040114	(200410)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
AU 2002242631	A1	20020919	(200433)		
ZA 2003007645	A	20050126	(200513)		311
NZ 528597	A	20050225	(200519)		
AU 2002242631	A8	20050915	(200569)		
IN 2003001594	P1	20051014	(200580)	EN	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002070721	A2	WO 2002-DK157	20020308
US 2003134353	A1 Provisional	US 2001-274650P	20010312
		US 2002-92947	20020308
EP 1379666	A2	EP 2002-708250	20020308
		WO 2002-DK157	20020308
AU 2002242631	A1	AU 2002-242631	20020308
ZA 2003007645	A	ZA 2003-7645	20030930
NZ 528597	A	NZ 2002-528597	20020308
		WO 2002-DK157	20020308
AU 2002242631	A8	AU 2002-242631	20020308
IN 2003001594	P1	WO 2002-DK157	20020308
		IN 2003-DN1594	20031006

FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 1379666	A2 Based on	WO 2002070721
AU 2002242631	A1 Based on	WO 2002070721

Searcher : Shears 571-272-2528

NZ 528597 A Based on WO 2002070721
AU 2002242631 A8 Based on WO 2002070721

PRIORITY APPLN. INFO: US 2001-274650P 20010312; DK 2001-395 20010308

AN 2002-723266 [78] WPIDS

AB WO 200270721 A UPAB: 20021204

NOVELTY - An isolated polynucleotide (I) comprising a nucleotide sequence encoding at least one regulator of morphology capable of regulating the morphology of a dimorphic fungal cell, and operably linked to a nucleotide sequence comprising an expression signal capable of directing the expression of the first sequence in a dimorphic fungal cell, where the sequences are not natively associated, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polypeptide capable of regulating the morphology of a dimorphic fungal cell and encoded by the first nucleotide sequence of (I);

(2) a extrachromosomal, recombinant DNA molecule, preferably in the form of an expression vector, comprising (I);

(3) a fungal host cell transfected or transformed with (I) or the vector of (2);

(4) a dimorphic fungal cell comprising:

(a) at least one nucleotide sequence encoding a gene product, and operably linked to, and

(b) at least one further nucleotide sequence comprising a further expression signal capable of directing the expression in a dimorphic fungal cell of the at least one nucleotide sequence encoding the gene product, where the further expression signal is regulatable, during growth of the dimorphic fungal cell, by one or more of:

(i) the composition of the growth medium, including at least one of carbon source, nitrogen source including amino acids or precursors, oxygen content, ionic strength, including NaCl content, pH, low molecular weight compounds, cAMP, and the presence or absence of a cell constituent, or its precursor;

(ii) the temperature of the growth medium, including any change thereof, including an upshift eliciting the expression of one or more heat shock genes;

(iii) the growth phase of the dimorphic fungal cell; and

(iv) the growth rate of the dimorphic fungal cell, where the nucleotide sequence encoding the gene product and the further nucleotide sequence comprising the regulatable expression signal are not natively associated;

(5) constructing a recombinant fungal cell of (3) or a recombinant dimorphic fungal cell of (4), the method comprising the step of transforming or transfecting (I) or the vector of (2) into a fungal cell or a dimorphic fungal cell;

(6) regulating the morphology of a recombinant fungal cell of (3) or a recombinant dimorphic fungal cell of (4), the method comprising the steps of cultivating the fungal cell or the dimorphic fungal cell under conditions allowing expression of the first nucleotide sequence encoding the at least one regulator of morphology, and regulating the morphology of the recombinant fungal cell or the recombinant dimorphic fungal cell;

(7) obtaining a predetermined dimorphic shift of a dimorphic fungal cell of (4), comprising cultivating the dimorphic fungal cell under conditions allowing expression of the first nucleotide sequence encoding the at least one regulator of morphology, and obtaining a

predetermined dimorphic shift of the dimorphic fungal cell, where the dimorphic shift results from regulating the expression in the dimorphic cell of the regulator of morphology;

(8) increasing the filamentation of a dimorphic fungal cell of (4), comprising cultivating the dimorphic fungal cell under conditions allowing expression of the first nucleotide sequence encoding the at least one regulator of morphology, and increasing the filamentation of the dimorphic fungal cell, where the increased filamentation results from regulating the expression in the dimorphic cell of the regulator of morphology;

(9) increasing the secretory capacity of a dimorphic fungal cell of (4), comprising cultivating the dimorphic fungal cell under conditions allowing expression of the first nucleotide sequence encoding the at least one regulator of morphology, and increasing the secretory capacity of the dimorphic fungal cell, where the increased secretory capacity results from regulating the expression in the dimorphic cell of the regulator of morphology; and

(10) producing a gene product in a dimorphic fungal cell of (4), comprising cultivating the dimorphic fungal cell under conditions allowing expression of the first nucleotide sequence encoding the at least one regulator of morphology, and cultivating the dimorphic fungal cell under conditions allowing expression of the nucleotide sequence encoding the gene product, and producing a gene product.

USE - The compositions and methods of the present invention are useful for increasing production and/or secretion of large quantities of commercially valuable proteins.

Dwg.0/23

L22 ANSWER 14 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-682760 [73] WPIDS
DOC. NO. CPI: C2002-192621
TITLE: New human, mouse or rat RET16 genes and proteins, involved in intracellular signaling cascade, useful for in gene therapy, particularly for treating e.g. ischemia, cystic fibrosis, autoimmune disease, cancers, tumors or neoplasms.
DERWENT CLASS: B04 D16
INVENTOR(S): FINGER, J; RILLEMA, J; TODDERUD, C G; FINGER, J N
PATENT ASSIGNEE(S): (FING-I) FINGER J; (RILL-I) RILLEMA J; (TODD-I) TODDERUD C G; (BRIM) BRISTOL-MYERS SQUIBB CO
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002066494	A2	20020829	(200273)*	EN	175
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW					
US 2002187492	A1	20021212	(200301)		
AU 2002250151	A1	20020904	(200427)		
JP 2004532619	W	20041028	(200471)		244
MX 2003007275	A1	20040101	(200471)		
EP 1575984	A2	20050921	(200562)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL					

PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2002066494	A2		WO 2002-US5162	20020215
US 2002187492	A1	Provisional	US 2001-269366P	20010216
		Provisional	US 2001-294181P	20010529
			US 2002-77111	20020215
AU 2002250151	A1		AU 2002-250151	20020215
JP 2004532619	W		JP 2002-566207	20020215
			WO 2002-US5162	20020215
MX 2003007275	A1		WO 2002-US5162	20020215
			MX 2003-7275	20030814
EP 1575984	A2		EP 2002-719045	20020215
			WO 2002-US5162	20020215

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2002250151	A1	Based on	WO 2002066494
JP 2004532619	W	Based on	WO 2002066494
MX 2003007275	A1	Based on	WO 2002066494
EP 1575984	A2	Based on	WO 2002066494

PRIORITY APPLN. INFO: US 2001-294181P 20010529; US 2001-269366P 20010216; US 2002-77111 20020215

AN 2002-682760 [73] WPIDS

AB WO 200266494 A UPAB: 20021113

NOVELTY - A new isolated polynucleotide (I) designated RET16 or its splice variant (RET16.2 or RET16.3), which encodes a cell signaling polypeptide involved in the intracellular signaling cascade, is new.

DETAILED DESCRIPTION - A new isolated polynucleotide designated RET16 or its splice variant (RET16.2 or RET16.3), which encodes a cell signaling polypeptide involved in the intracellular signaling cascade, is new. The RET16 polynucleotide comprises 1818, 1553, 630, 1901 or 520 base pairs fully defined in the specification. The RET polynucleotide encodes a polypeptide with the sequence having 476, 512, 475 or 71 amino acids also fully defined in the specification. The nucleic acid sequence has the ATCC Accession Number PTA-3161.

INDEPENDENT CLAIMS are also included for the following:

(1) a composition (II) comprising the RET16 polynucleotide;

(2) an expression vector (III) or recombinant vector containing (I);

(3) (recombinant) host cells (IV) containing the vector;

(4) a substantially purified cell signaling protein (V) encoded by (I);

(5) a purified antibody (V), which binds specifically to the protein or polypeptide, or its antigenic epitope;

(6) producing (M1) a protein involved in the cell signaling cascade;

(7) detecting (M2) a polynucleotide encoding a cell signaling cascade protein or its fragment in a biological sample;

(8) screening (M3) a library of molecules or compounds with a polynucleotide encoding a protein involved in the cell signaling cascade to identify at least one molecule or compound that

specifically binds to the polynucleotide sequence;

(9) screening (M4) for candidate compounds capable of modulating activity of a cell signaling protein involved in the cell signaling cascade;

(10) screening (M5) for compounds that inhibit or prevent binding of a human cell signaling protein with a second cell signaling protein;

(11) identifying (M6) compounds that inhibit the phosphorylation of a cell signaling cascade protein by protein kinases; and

(12) an antisense RET16 oligonucleotide (VI) that is complementary to the RET16 polynucleotide sequences of hRET16a, human RET16.2 splice variant DNA, or human RET16.3 splice variant DNA.

ACTIVITY - Anti-inflammatory; Cytostatic; Vasotropic; Neuroprotective; Antiasthmatic; Immunosuppressive; Cardiovascular; Antiarthritic; Antiallergic; Cardiant; Antidiabetic.

No biological data given.

MECHANISM OF ACTION - Gene therapy; RET16 modulator.

USE - The RET16 protein or polynucleotide is useful for treating an inflammation-related disease or disorder, e.g. rheumatoid arthritis, juvenile arthritis, psoriasis, asthma, ischemia-reperfusion, multiple sclerosis, rejection of organ or tissue transplants, chronic obstructive pulmonary disease, inflammatory bowel disease, Crohn's disease, ulcerative colitis, inacute respiratory distress syndrome, systemic lupus erythematosus, cystic fibrosis, autoimmune disease, cancers, tumors or neoplasms. This inflammation-related disease or disorder also includes disorders associated with aberrant activation of the TNF- alpha pathway, disorders associated with aberrant cellular migration, disorders associated with aberrant cellular proliferation, disorders associated with aberrant cellular metastasis, juvenile idiopathic arthritis, hematogenous metastases of tumor cells, hyperinsulinemia, diabetes type 2, atherosclerosis, cardiovascular disease, tumor progression, metastasis, colon cancer, Wegener's granulomatosis, stem cell transplantation complications, thalassemia, autoimmune disease, ischemia-reperfusion injury, acute lung injury, graft rejection, systemic lupus, coronary artery calcification, ischemic heart, or allergic inflammation (all claimed). These polypeptides and polynucleotides are especially useful in gene therapy. The modulators of RET16 are also useful for treating the above-mentioned diseases.

Dwg.0/21

L22 ANSWER 15 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER:	2002-490152 [52] WPIDS
DOC. NO. NON-CPI:	N2002-387450
DOC. NO. CPI:	C2002-139210
TITLE:	Evaluating effect of drugs on nervous system by comparing effect of drug on acetylcholinesterase, AChE activity in brain of test animal following challenge by AChE blocker and comparing it with control group.
DERWENT CLASS:	B04 D16 S03
INVENTOR(S):	MESHORER, E; SHOHAM, S; SKLAN, E; SOREG, H; SOREQ, H
PATENT ASSIGNEE(S):	(YISS) YISSUM RES DEV CO HEBREW UNIV JERUSALEM; (MESH-I) MESHORER E; (SHOH-I) SHOHAM S; (SKLA-I) SKLAN E; (SORE-I) SOREG H
COUNTRY COUNT:	98

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

WO 2002040994 A2 20020523 (200252)* EN 114
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2002023996 A 20020527 (200261)
US 2004058357 A1 20040325 (200422)
AU 2002223996 A8 20051006 (200612)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002040994	A2	WO 2001-IL1051	20011114
AU 2002023996	A	AU 2002-23996	20011114
US 2004058357	A1	WO 2001-IL1051	20011114
		US 2003-432131	20030926
AU 2002223996	A8	AU 2002-223996	20011114

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002023996	A Based on	WO 2002040994
AU 2002223996	A8 Based on	WO 2002040994

PRIORITY APPLN. INFO: US 2000-247970P 20001114; US 2003-432131 20030926

AN 2002-490152 [52] WPIDS

AB WO 200240994 A UPAB: 20020815

NOVELTY - Evaluating (M) an effect on the nervous system of a test drug, comprises comparing the effect of the drug on acetylcholinesterase (AChE) catalytic activity or isoform variance in a brain of a test animal following a challenge by an AChE blocker or a blocker of AChE and muscarinic receptors M1 and M2 and comparing with a control group tested under the same conditions but not treated with the drug.

DETAILED DESCRIPTION - Evaluating (M) an effect on the nervous system of a test drug comprises:

(a) providing a control group and a test group;

(b) at time zero, injecting each of the animals of the control group with a carrier and each of the animals of the test group with the test drug contained within the carrier;

(c) after a predetermined period of time from time zero, injecting animals of the control group with a carrier, an irreversible acetylcholinesterase (AChE) blocker or an AChE, M1 and M2 blocker, and injecting the animals of the test group with the same agent used for the control group;

(d) after a first predetermined period of time from zero time sacrificing a number of animals from each of the control and test groups and dissecting their brains, sacrificing the remaining of animals after a second predetermined period of time;

(e) subjecting the dissected brains to an analytical procedure for assessing AChE catalytic activity or isoform variance, or their combinations;

(f) generating a drug profile from the results of the analytical procedures performed in each of the control and test groups; and

(g) comparing the profile of the test group with the profile displayed by the control groups, where increased expression in the brain of AChE mRNA transcripts, with or without a shift in the alternative splicing of the transcripts from the primary S to the normally rare R variants indicates a distinct feed back response to the test drug, an increase with time in the catalytic activity of brain AChE, with or without an increase in the globular G1 AChE monomers to G4 tetramers reflects the outcome of such feed back at protein level, and any diversion from the brain response of the control to the irrevasible AChE blocker reflects an impairment in muscarinic neurotransmission resulting from the exposure to the test drug.

An INDEPENDENT CLAIM is also included for a system for assaying a drug for its effect on the central nervous system, comprising groups of test animals including one treated control group and an identical number of corresponding test groups, a carrier, a general non-selective muscarinic receptor blocker in the carrier, a selective muscarinic receptor 1 (M1) blocker in the carrier and unit for injecting the same into the animals, an AChE blocker and unit for injecting the same into the animals, AChE, M1 and M2 blocker and unit for injecting the same into the animals, unit for sacrificing the animals and for dissecting their brains, and unit for assessing AChE catalytic activity or AChE isoform variance, or their combinations in the dissected brains.

ACTIVITY - Nootropic; Neuroprotective; Muscular; Tranquilizer; Antiparkinsonian. No suitable biological data is given.

MECHANISM OF ACTION - AChE expression modulator.

USE - (M) is useful for evaluating an effect on the nervous system of a test drug including drugs for the treatment of anxiety conditions, post-traumatic stress, Alzheimer's disease, muscle malfunctioning, neurodegenerative disorders, damage resulting from exposure to xenobiotics, panic, neuromuscular disorders, Parkinson's disease, Huntington's chorea, muscle fatigue, multiple chemical sensitivity, autism, multiple sclerosis and Shorgren's disease (claimed).

Dwg.0/22

L22 ANSWER 16 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-315802 [35] WPIDS
DOC. NO. NON-CPI: N2002-247104
DOC. NO. CPI: C2002-092030
TITLE: New DSP-16 polypeptide, useful for identifying modulators of its activity, which can be used in the treatment of disorders such as Duchenne muscular dystrophy, or cancer.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): LUCHE, R M; WEI, B
PATENT ASSIGNEE(S): (CEPT-N) CEPTYR INC
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002026997	A2	20020404	(200235)*	EN	87

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE

DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG
KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH
PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU
ZA ZW

AU 2001094744 A 20020408 (200252)
US 2002137170 A1 20020926 (200265)
AU 2001294744 A8 20051006 (200612)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002026997	A2	WO 2001-US30124	20010925
AU 2001094744	A	AU 2001-94744	20010925
US 2002137170	A1 Provisional	US 2000-235487P	20000926
		US 2001-964277	20010925
AU 2001294744	A8	AU 2001-294744	20010925

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001094744	A Based on	WO 2002026997
AU 2001294744	A8 Based on	WO 2002026997

PRIORITY APPLN. INFO: US 2000-235487P 20000926; US 2001-964277 20010925

AN 2002-315802 [35] WPIDS

AB WO 200226997 A UPAB: 20020603

NOVELTY - An isolated **polypeptide** having a 665 amino acid DSP-16 sequence (S1), given in the specification, or a variant having at least 50 % identical residues, which retains the ability to dephosphorylate an activated mitogen-activated **protein kinase**, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated **polypeptide** a DSP-16 alternate form sequence, having a 517 amino acid sequence, (S5), given in the specification;

(2) an isolated polynucleotide encoding at least ten, preferably at least 15 consecutive amino acids of (S1) or (S5);

(3) a polynucleotide comprising a 3496 nucleotide sequence (S2), given in the specification;

(4) an antisense polynucleotide comprising at least 15 consecutive nucleotides complementary to (S2);

(5) an isolated polynucleotide that detectably **hybridizes** to the complement of (S1) under conditions including a wash in 0.1x **saline sodium citrate** (SSC) and 0.1 % sodium dodecyl sulfate (SDS) at 50 deg. C for 15 minutes;

(6) an expression vector comprising the polynucleotide of (2), (3), (4) or (5);

(7) a host cell transformed or transfected with a vector of (6);

(8) producing a DSP-16 **polypeptide**, comprising:

(a) culturing the vector of (6) containing the polynucleotide of (3) under expression conditions; and

(b) isolating DSP-16 **polypeptide**;

(9) an antibody specific for (S1);

(10) detecting DSP-16 expression in a sample, comprising:

(a) contacting a sample with the antibody of (9); and

(b) detecting the level of antibody/DSP-16 complex;
(11) detecting DSP-16 expression in a sample, comprising:
(a) contacting the sample with the sequence of (4); and
(b) detecting the amount of DSP-16 polynucleotide that **hybridizes** to the antisense polynucleotide;
(12) screening for an agent that modulates DSP-16 activity, comprising:
(a) contacting a candidate agent with the novel **polypeptide**, under interacting conditions; and
(b) evaluating the ability of the **polypeptide** to dephosphorylate a DSP-16 substrate relative to a control;
(13) screening for an agent that modulates DSP-16 activity, comprising:
(a) contacting a candidate agent with a cell comprising a DSP-16 promoter operably linked to a polynucleotide encoding a detectable transcript or **protein**, under interaction conditions; and
(b) evaluating the expression of the polynucleotide relative to a predetermined level of expression in the absence of the agent; and
(14) a DSP-16 substrate trapping mutant that differs from (S1) at upto 50 % of the residues, and retains the same substrate affinity, but has reduced dephosphorylation activity.
ACTIVITY - Inotropic; Cytostatic; Immunosuppressive; Antiallergic.
No biological data is given.
MECHANISM OF ACTION - DSP-16 modulator.
USE - For identifying agents which modulate DSP-16 activity, for modulation of a proliferative response in a cell, survival of a cell, or differentiation of a cell. The cell displays contact inhibition of cell growth or anchorage independent growth. The cell may display altered intercellular adhesion. The agent may modulate apoptosis, or the cell cycle. The identified modulators can be used to treat Duchenne muscular dystrophy, cancer, graft-versus-host disease, autoimmune diseases, allergies, metabolic diseases, abnormal cell growth, abnormal cell proliferation, and cell cycle abnormalities. (All claimed).
Dwg.0/5

L22 ANSWER 17 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-351892 [38] WPIDS
DOC. NO. CPI: C2002-099994
TITLE: New **protein kinase** B, pknB gene from corynebacteria, useful as hybridization probe and overexpression of which gene in corynebacteria is useful for producing L-amino acids, in particular L-lysine.
DERWENT CLASS: B04 D13 D16
INVENTOR(S): BATHE, B; FARWICK, M; HANS, S; HERMANN, T
PATENT ASSIGNEE(S): (DEGS) DEGUSSA AG
COUNTRY COUNT: 98
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002022828	A1	20020321	(200238)*	EN	46

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH

PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

DE 10120095	A1	20020328	(200238)
US 2002042105	A1	20020411	(200238)
AU 2001082132	A	20020326	(200251)
EP 1317547	A1	20030611	(200339) EN

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

US 2005130277	A1	20050616	(200540)
US 6939692	B2	20050906	(200558)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002022828	A1	WO 2001-EP10211	20010905
DE 10120095	A1	DE 2001-10120095	20010425
US 2002042105	A1 Provisional	US 2001-297250P	20010612
		US 2001-949970	20010912
AU 2001082132	A	AU 2001-82132	20010905
EP 1317547	A1	EP 2001-960723	20010905
		WO 2001-EP10211	20010905
US 2005130277	A1 Provisional	US 2001-297250P	20010612
	Div ex	US 2001-949970	20010912
		US 2004-940606	20040915
US 6939692	B2 Provisional	US 2001-297250P	20010612
		US 2001-949970	20010912

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001082132	A Based on	WO 2002022828
EP 1317547	A1 Based on	WO 2002022828

PRIORITY APPLN. INFO: DE 2001-10120095 20010425; DE 2000-10044912 20000912

AN 2002-351892 [38] WPIDS

AB WO 200222828 A UPAB: 20020618

NOVELTY - An isolated polynucleotide (PN) (I) from corynebacteria, comprising a PN sequence coding for pknB gene, consisting of a PN 70% identical to a PN which codes for a polypeptide comprising a sequence (S1) of 627 amino acids given in specification, or a PN which codes for a polypeptide comprising a sequence 70% identical to (S1), the polypeptide preferably having activity of **protein kinase** B, is new.

DETAILED DESCRIPTION - An isolated polynucleotide (PN) (I) from corynebacteria is chosen from:

(a) a PN which has a sequence 70% identical to a PN which codes for a polypeptide comprising a fully defined sequence (S1) of 627 amino acids as given in the specification;

(b) a PN which codes for a polypeptide comprising a sequence 70% identical to (S1);

(c) a PN which is complementary to (a) or (b); and

(d) a PN comprising at least 15 successive nucleotides of (a), (b) or (c), is new.

INDEPENDENT CLAIMS are also included for the following:

Corynebacterium glutamicum DM1547 deposited as DSM13994 in the German Collection of Microorganisms and Cell Cultures;

(1) corynebacteria (II), in which the pknB gene is amplified, and in particular, overexpressed;

(2) a DNA (III) originating from corynebacteria and coding for **protein kinase** B, where the corresponding amino acid sequences:

(a) between positions 581-587 in (S1) are modified by amino acid exchange;

(b) contain any other proteogenic amino acid except L-proline in position 584 in (S1);

(c) contains L-serine or L-threonine in position 584 in (S1); or

(d) contains L-serine at position 584 in a sequence of 627 amino acids;

(3) a corynebacteria containing (III) or a vector carrying (I);

(4) detecting RNA, cDNA and DNA in order to isolate nucleic acids or PN or genes which code for pknB or have a high degree of similarity to the sequence of the pknB gene, where (I) are used as hybridization probes; and

(5) a fermentation process for the preparation of L-amino acids, especially L-lysine.

USE - (I) is useful as hybridization probe for discovering RNA, cDNA and DNA to isolate nucleic acids, or PNs or genes which code for **protein kinase** B or have a high similarity with the sequence of the pknB gene. The method employs arrays, microarrays or DNA chips. (II) is useful for preparing L-amino acids, in particular L-lysine, by fermenting (II), in particular strain C. glutamicum strain DSM 13994, concentrating the desired product in the medium or in the cells of the bacteria, and isolating the L-amino acid. In this method, bacteria in which further genes of the biosynthesis pathway of the desired L-amino acid are additionally amplified, are employed. Bacteria employed are those in which the metabolic pathways which reduce the formation of desired L-amino acids are at least partially switched off. A strain transformed with a plasmid vector is used and the vector carries the nucleotide sequence coding for the pknB gene. The expression of the PNs coding for the pknB gene is amplified, and in particular overexpressed. The catalytic properties of the polypeptide for which the PN pknB codes are enhanced. For the preparation of L-amino acids, in particular L-lysine, bacteria are fermented, in which at the same time one or more of endogenous genes such as the dapA gene which codes for dihydrodipicolinate synthase, the gap gene which codes for glyceraldehyde 3-phosphate dehydrogenase, the tpi gene coding triose phosphate isomerase, the pgk gene coding for 3-phosphoglycerate kinase, the zwf gene which codes for glucose 6-phosphate dehydrogenase, the pyc gene which codes for pyruvate carboxylase, the lysE gene which codes for lysine export, the lysC gene which codes for a feed back resistant aspartate kinase, hom gene coding for homoserine dehydrogenase, ilvA gene coding for threonine dehydratase or the ilvA(Fbr) allele which codes for a feed back resistant threonine dehydratase, the ilvBN gene coding for acetohydroxy-acid synthase, the ilvD gene coding for dihydroxy-acid dehydratase, the zwa1 gene which codes for the Zwa1 protein, is or are enhanced, preferably over-expressed. Further the bacteria has attenuation in one or more genes such as the pck gene which codes for phosphoenol pyruvate carboxykinase, the pgi gene which codes for glucose 6-phosphate isomerase, the poxB gene which codes for pyruvate oxidase, and the zwa2 gene which codes for the Zwa2 protein (all claimed). The L-amino acids are useful in the food industry and in animal nutrition.

ADVANTAGE - Corynebacterium produces amino acids in an improved manner after attenuation of the pknB gene.

Dwg.0/0

L22 ANSWER 18 OF 39 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:597071 BIOSIS
DOCUMENT NUMBER: PREV200200597071
TITLE: Characterization of the Cryptococcus neoformans Mitogen-activated Protein kinase CnHOG1.
AUTHOR(S): Saha, S. K. [Reprint author]; Chaturvedi, V. [Reprint author]
CORPORATE SOURCE: Wadsworth Center, NY State Dept. of Health, Albany, NY, USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2002) Vol. 102, pp. 204. print.
Meeting Info.: 102nd General Meeting of the American Society for Microbiology. Salt Lake City, UT, USA. May 19-23, 2002. American Society for Microbiology.
ISSN: 1060-2011.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Nov 2002
Last Updated on STN: 20 Nov 2002

AB Background: Mitogen-activated **Protein** (MAP) cascades are crucial in determining cellular response to external stimuli. These genes are broadly conserved from yeast to mammals. Saccharomyces cerevisiae homologs that transduce signals for mating and nitrogen starvation are reported to be critical for regulation of pathogenesis in Cryptococcus neoformans. S. cerevisiae also has a well-defined High Osmolarity Glycerol (HOG) pathway for sensing osmotic stress in the environment. We are studying this pathway to understand how C. neoformans responds to stress and if components of this pathway have a role in fungal pathogenesis. Methods: A C. neoformans yeast expression library previously reported from our laboratory, was screened to complement salt sensitivity of a S. cerevisiae hog1 mutant. Plasmid preparation, extraction, restriction digestion, Southern and northern blotting, and nucleotide sequencing were according to standard procedures. Yeast transformation was done with Frozen-E Z Yeast transformation kit. Results: 2X106 yeast transformants carrying entire C. neoformans cDNA, were spread on YPD-0.4 M **NaCl** plates. One positive clone that conferred salt resistance was recovered, and the plasmid was isolated, and its ability to confer desired phenotype was re-confirmed. The positive plasmid contained a 1.2 kb insert. Sequence analysis showed ORF of 365 aa with a putative conserved **protein kinase** domain. Database searches revealed close identity with HOG1p from Schizosaccharomyces pombe (78%), S. cerevisiae (74%), and Candida albicans (70%) and a more distant identity with Human MAP kinase P38 (47%). Genomic Southern blots revealed one copy of CnHOG1. A 1.1. kb partial genomic clone was identified by cross-**hybridization** with cDNA and sequence comparison using gap function of GCG software showed two introns. C. neoformans cells exposed to high salt showed activation of HOG1 transcript in northern blot experiments. Gene disruption and pathogenesis experiments are in progress. Conclusion: We have characterized a C. neoformans (CnHOG1) homolog of yeast MAP kinase HOG1, which is involved in response to osmotic stress.

L22 ANSWER 19 OF 39 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation

on STN
ACCESSION NUMBER: 2003:326325 BIOSIS
DOCUMENT NUMBER: PREV200300326325
TITLE: VALPROIC ACID ALTERS THE EXPRESSION OF GENES ENCODING VOLTAGE-GATED SODIUM CHANNELS IN THE RAT BRAIN.
AUTHOR(S): Burnet, P. W. J. [Reprint Author]; Pei, Q.; Hutchinson, L. [Reprint Author]; Harrison, P. J. [Reprint Author]
CORPORATE SOURCE: Psychiatry, Oxford University, Oxford, UK
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 743.11. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

AB Valproic acid (or 'valproate') is an anticonvulsant which is used as a 'mood stabilizer' in the treatment of bipolar disorder. It has been suggested that valproate exerts its therapeutic effects by impairing neuronal sodium ion currents and thus, the excessive accumulation of intracellular sodium. This effect might be mediated through **protein kinase** C (PKC), which is inhibited by valproate, and which influences the expression of sodium channel (NaV1.1) alpha-1 and beta-1 subunit mRNAs in vitro. We therefore, investigated whether valproate alters the expression of voltage-gated sodium channel subunit mRNAs in the rat brain, as predicted by the hypothesis that the mood stabilizer modulates sodium ion currents in neurons. Immunoautoradiography, using specific antibodies, was used to evaluate the concentration of sodium channel subunit **proteins** in the brains of rats treated with valproate for fourteen days at standard doses. The expression of subunit mRNAs in the same animals was estimated using in situ **hybridization** histochemistry. Valproate decreased the abundance of NaV1.1 mRNA (-28%, $p<0.05$) and **protein** (-24%, $p<0.05$) in the parietal cortex compared to **saline** controls. However, the expression of NaV1.2 and NaV1.6 mRNAs was not affected by valproate. Measurements of **protein** are ongoing. The data, therefore, demonstrate that valproate alters sodium channel gene expression and suggests that neuronal activity might be altered. The reduction in the abundance of mRNAs which are used as proxies for neuronal activity by valproate, supports this conclusion.

L22 ANSWER 20 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2001-565793 [63] WPIDS
CROSS REFERENCE: 2003-636734 [60]
DOC. NO. CPI: C2001-167950
TITLE: Attenuating gene expression in a cell using gene-targeted double stranded RNA.
DERWENT CLASS: B04 D16 P13 P14
INVENTOR(S): BEACH, D; BERNSTEIN, E; CAUDY, A; HAMMOND, S; HANNON, G; BEACH, D H; CONKLIN, D; HANNON, G J; PADDISON, P J
PATENT ASSIGNEE(S): (COLD-N) COLD SPRING HARBOR LAB; (GENE-N) GENETICA INC; (BEAC-I) BEACH D; (BERN-I) BERNSTEIN E; (CAUD-I) CAUDY A; (HAMM-I) HAMMOND S; (HANN-I) HANNON G
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001068836	A2	20010920	(200163)*	EN	134
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001045793	A	20010924	(200208)		
US 2002162126	A1	20021031	(200274)		
EP 1272630	A2	20030108	(200311)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
US 2003084471	A1	20030501	(200331)		
JP 2003526367	W	20030909	(200360)		149
US 2004018999	A1	20040129	(200413)		
US 2004086884	A1	20040506	(200430)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001068836	A2		WO 2001-US8435	20010316
AU 2001045793	A		AU 2001-45793	20010316
US 2002162126	A1	Provisional	US 2000-189739P	20000316
		Provisional	US 2000-243097P	20001024
		CIP of	WO 2001-US8435	20010316
			US 2001-866557	20010524
EP 1272630	A2		EP 2001-918752	20010316
			WO 2001-US8435	20010316
US 2003084471	A1	Provisional	US 2000-189739P	20000316
		Provisional	US 2000-243097P	20001024
		CIP of	WO 2001-US8435	20010316
			US 2002-55797	20020122
JP 2003526367	W		JP 2001-567320	20010316
			WO 2001-US8435	20010316
US 2004018999	A1	Provisional	US 2000-189739P	20000316
		Provisional	US 2000-243097P	20001024
		CIP of	WO 2001-US8435	20010316
			US 2001-858862	20010516
US 2004086884	A1	Provisional	US 2000-189739P	20000316
		Provisional	US 2000-243097P	20001024
		CIP of	WO 2001-US8435	20010316
		CIP of	US 2001-858862	20010516
		CIP of	US 2001-866557	20010524
		CIP of	US 2002-55797	20020122
			US 2003-350798	20030124

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001045793	A	Based on	WO 2001068836
EP 1272630	A2	Based on	WO 2001068836
JP 2003526367	W	Based on	WO 2001068836

PRIORITY APPLN. INFO: US 2000-243097P 20001024; US

2000-189739P 20000316; US
2001-866557 20010524; US
2002-55797 20020122; US
2001-858862 20010516; US
2003-350798 20030124

AN 2001-565793 [63] WPIDS
CR 2003-636734 [60]
AB WO 200168836 A UPAB: 20040511
NOVELTY - Methods for attenuating gene expression in a cell using gene-targeted double stranded RNA (dsRNA), are new. The dsRNA comprises a sequence that hybridizes to the sequence of the gene to be inhibited (the target).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a method (I) for attenuating expression of a target gene in a non-embryonic cell suspended in culture, comprising introducing a double stranded RNA (dsRNA) into the cell to attenuate expression of the target gene (the dsRNA comprises a nucleotide sequence that hybridizes under stringent conditions to a nucleotide sequence of the target gene);

(2) an assay (II) for identifying nucleic acid sequences responsible for conferring a particular phenotype in a cell, comprising:

(a) constructing a variegated library of nucleic acid sequences from a cell in an orientation relative to a promoter to produce dsDNA;

(b) introducing the variegated dsRNA library into a culture of target cells which have an activated Dicer activity or Argonaut activity; and

(c) identifying members of the library which confer a particular phenotype on the cell and identifying the sequence from a cell which correspond. e.g. being identical or homologous, to the library member;

(3) a method (III) of conducting a drug discovery business, comprising:

(a) identifying, via (II), a target gene which provides a phenotypically desirable response when inhibited by RNA;

(b) identifying agents by their ability to inhibit expression of the target gene or the activity of an expression product of a target gene;

(c) conducting therapeutic profiling of agents identified by step (b), or further analogs of them, for efficacy and/or toxicity in animals; and

(d) formulating a pharmaceutical preparation comprising the agents identified in step (c) as having an acceptable therapeutic profile; and

(4) a method (IV) of conducting a target discovery business, comprising:

(a) identifying, via (II), a target gene which provides a phenotypically desirable response when inhibited by RNA;

(b) (optionally) conducting therapeutic profiling of the target gene for efficacy and toxicity in animals; and

(c) licensing, to a third party, the rights for further drug development of the inhibitors of the target gene.

USE - The methods are used for attenuating target gene expression in non-embryonic cells suspended in culture or in animals.
Dwg.0/27

L22 ANSWER 21 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2001-138148 [14] WPIDS
CROSS REFERENCE: 2000-665011 [60]; 2001-138149 [09]

DOC. NO. NON-CPI: N2001-100576
DOC. NO. CPI: C2001-040690
TITLE: New dual-specificity phosphatase-3 polypeptide and its variants useful for treating disorders associated with DSP-3 activity, defects in cell proliferation, differentiation or survival, e.g. Duchenne muscular dystrophy, cancer.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): LUCHE, R M; WEI, B
PATENT ASSIGNEE(S): (CEPT-N) CEPTYR INC
COUNTRY COUNT: 92
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001002581	A1	20010111	(200114)*	EN	70

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000043676	A	20010122 (200125)	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001002581	A1	WO 2000-US10868	20000420
AU 2000043676	A	AU 2000-43676	20000420

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000043676	A	Based on	WO 2001002581

PRIORITY APPLN. INFO: WO 2000-US9185 20000407; US 1999-142338P 19990702
AN 2001-138148 [14] WPIDS
CR 2000-665011 [60]; 2001-138149 [09]
AB WO 200102581 A UPAB: 20010508
NOVELTY - An isolated **polypeptide** having the 184 amino acid dual-specificity phosphatase-3 (DSP-3) sequence (I) fully defined in the specification, or its variant, is new. The variant has deletions, additions, insertions or substitutions at no more than 50 % of the residues of (I), and retains the ability to dephosphorylate an activated MAP-kinase.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide that encodes at least 10 or 15 consecutive amino acids of (I);

(2) expression vectors comprising the polynucleotides of (1);

(3) host cells transformed or transfected with the vectors of (2);

(4) an isolated polynucleotide encoding (I);

(5) an antisense polynucleotide comprising at least 15 consecutive nucleotides complementary to the polynucleotide of (4);

(6) an isolated polynucleotide that detectably **hybridizes**

to the complement of a 926 base pair sequence (II) fully defined in the specification, under conditions that include a wash in 0.1 multiply SSC (**saline sodium chloride**) and 0.1 % sodium dodecyl sulfate (SDS) at 50 deg. C for 15 minutes;

(7) a method of producing a DSP-3 **polypeptide** by culturing a host cell of (3) under expression conditions and isolating the **polypeptide** from the cell culture;

(8) an isolated antibody or its antigen binding fragment specifically binding to DSP-3, and a composition comprising the antibody or its antigen binding fragment and a carrier;

(9) detecting DSP-3 expression in a sample by contacting a sample with an antibody or antigen-binding fragment, or with an antisense polynucleotide, and detecting the level of antibody/DSP-3 complex or the amount of DSP-3 **hybridized** to the antisense polynucleotide, to detect DSP-3 expression;

(10) screening for an agent that modulates DSP-3 activity, comprising:

(a) contacting a candidate agent with (I) or a cell comprising a DSP-3 promoter operably linked to a polynucleotide encoding a detectable transcript or **protein**; and

(b) evaluating the ability of the **polypeptide** to dephosphorylate a DSP-3 substrate relative to a predetermined ability of the **polypeptide** to dephosphorylate the DSP-3 substrate in the absence of candidate; or

(c) evaluating the expression of the polynucleotide relative to a predetermined level of expression in the absence of a candidate agent;

(11) modulating a proliferative response, differentiation and survival of a cell by contacting a cell with an agent that modulates DSP-3 activity;

(12) a method for treating a patient afflicted with a disorder associated with DSP-3 activity by administering to a patient an agent that modulates DSP-3 activity;

(13) a DSP-3 substrate trapping mutant **polypeptide** that differs from (I) by deletions, additions, insertions or substitutions at no more than 50 % of the residues, it binds to a substrate with an affinity that is not substantially diminished relative to DSP-3, and the ability of the **polypeptide** to dephosphorylate a substrate is reduced relative to DSP-3; and

(14) screening a molecule for the ability to interact with DSP-3 by contacting a candidate molecule with (I) under interacting conditions, and detecting the presence of binding of the candidate molecule to (I).

ACTIVITY - Cytostatic; immunosuppressive; inotropic.

No biological data is given.

MECHANISM OF ACTION - Mitogen-activated **protein kinase** regulator.

USE - The DSP-3 **polypeptides** and agents that modulate DSP-3 activity are useful for treating conditions or disorders associated with DSP-3 activity, defects in cell proliferation, differentiation and/or survival. The conditions include Duchenne muscular dystrophy, cancer, graft-versus-host disease, autoimmune diseases, metabolic diseases, abnormal cell growth or proliferation, and cell cycle abnormalities. DSP-3 **polypeptides** may also be used to identify antibodies or other molecules that modulate signal transduction leading to proliferative responses, cell differentiation and/or survival, for immunization, and to identify modulators of DSP-3 activity.

Dwg.0/4

L22 ANSWER 22 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2001-138130 [14] WPIDS
DOC. NO. CPI: C2001-040672
TITLE: Novel polynucleotide encoding abscisic acid-activated **protein kinase** useful for regulating gas exchange and transpirational water loss in plants possessing stomata.
DERWENT CLASS: C06 D16
INVENTOR(S): ASSMANN, S M; LI, J
PATENT ASSIGNEE(S): (PENN-N) PENN STATE RES FOUND
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001002541	A2	20010111	(200114)*	EN	56

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000060604	A	20010122 (200125)	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001002541	A2	WO 2000-US18014	20000629
AU 2000060604	A	AU 2000-60604	20000629

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000060604	A	Based on	WO 2001002541

PRIORITY APPLN. INFO: US 2000-192499P 20000328; US 1999-142039P 19990701; US 2000-176245P 20000114

AN 2001-138130 [14] WPIDS

AB WO 200102541 A UPAB: 20010312
NOVELTY - An isolated nucleic acid molecule (I) encoding an abscisic acid (ABA)-activated **protein kinase** (AAPK), expressed predominantly in guard cells of a plant, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an oligonucleotide (II) greater than 15 nucleotides in length, which hybridizes with either strand of (I) under predetermined hybridization conditions;

(2) a polypeptide (III) produced by expression of (I);

(3) an antibody (IV) immunologically specific for (III);

(4) a vector (V) for transforming a plant cell, comprising (I);

(5) a transformed plant cell comprising (V);

(6) a genetically altered plant (VI) possessing decreased or increased sensitivity to ABA-induced stomatal closure as compared with an equivalent but unaltered plant, comprising an AAPK that is largely non-functional or absent or that is increased in amount or activity as compared with the unaltered plant;

(7) increasing transpiration in a plant comprising reducing or preventing function of an AAPK in guard cells of the plant which reduces sensitivity of the plant to ABA-induced stomatal closure;

(8) decreasing transpiration in a plant comprising increasing the amount or activity of an AAPK in guard cells of the plant which increases the sensitivity of the plant to ABA-induced stomatal closure; and

(9) a fertile plant with increased or decreased transpiration;

USE - (I) is useful for increasing or decreasing transcription in a plant (claimed). AAPK function is reduced, prevented or increased by the addition of (I) to the plant genome. (I) is useful for isolating or creating AAPK mutants in a selected species and as probes to detect the presence and/or expression of AAPK genes or to identify related genes from other plant species. (I) is useful for isolating and identifying other putative members of AAPK, in vivo. (I) can be used in crop and horticulture varieties such as corn, wheat, rye, oats, barley, rice, soybeans etc., in which reduction of moisture content is important. (I) is useful for accelerating or controlling the rate of crop drying. (I) has myriad applications to important plant problems especially in irrigated crops or other crops where water and yield are delicately balanced. (I) is also useful for a variety of agronomic purposes, such as to help enhance tolerance to water stress or drought conditions. (I) is useful to minimize water loss in fruits, vegetables and flowers, including cut flowers, during harvest, transport and distribution and increases the shelf-life and freshness of the fruits, vegetables and flowers. A polypeptide (III) encoded by (I) is useful to identify molecules with binding affinity for AAPK. An antibody (IV) specific to (III) is useful in affinity chromatography to isolate (III), to identify or quantify (III) or to immunoprecipitate (III) from a sample containing a mixture of proteins and other biological materials.

ADVANTAGE - (I) is of tremendous value to plant growers to accelerate or control the rate of crop drying. (I) extends shelf-life and maintains the freshness of fruits, vegetables and flowers by minimizing water loss during harvest, transport and distribution.

Dwg.0/4

L22 ANSWER 23 OF 39 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 2001186339 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11264326
TITLE: Limbic-cortical-ventral striatal activation during retrieval of a discrete cocaine-associated stimulus: a cellular imaging study with gamma protein kinase C expression.
AUTHOR: Thomas K L; Everitt B J
CORPORATE SOURCE: Department of Experimental Psychology, University of Cambridge, Cambridge CB2 3EB, United Kingdom.. klt25@cus.cam.ac.uk
SOURCE: The Journal of neuroscience : the official journal of the Society for Neuroscience, (2001 Apr 1) Vol. 21, No. 7, pp. 2526-35.
Journal code: 8102140. E-ISSN: 1529-2401.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010702
Last Updated on STN: 20010702

Searcher : Shears 571-272-2528

Entered Medline: 20010628

AB We investigated the neuronal activation associated with reexposure to a discrete cocaine-associated stimulus using in situ **hybridization** to quantify the expression of the plasticity-regulated gene, gamma **protein kinase** C (gamma PKC), in the limbic-cortical-ventral striatal system. Groups of rats were trained to self-administer cocaine paired with a light stimulus (Paired) or paired with an auditory stimulus but also receiving light presentations yoked to those in the Paired group (Unpaired). Additional groups received noncontingent cocaine-light pairings (Pavlovian) or **saline**-light pairings (**Saline**) that were yoked to the Paired group. After acquisition of self-administration by the Paired and Unpaired groups, all groups had a 3 d drug- and training-free period before being reexposed to noncontingent presentations of the light conditioning stimulus during a 5 min test session in the training context. There were four major patterns of results for regional gamma PKC expression 2 hr later. (1) Changes occurred only in groups in which the light was predictive of cocaine. (2) Increases were seen in the amygdala, but decreases were seen in the medial prefrontal cortex. (3) No changes were seen in the hippocampus. (4) Although changes were observed in the basal and central nuclei of the amygdala and the prelimbic cortex in both the Paired and Pavlovian groups, additional changes were observed in the nucleus accumbens core, lateral amygdala, and anterior cingulate cortex in the Pavlovian group. These results suggest not only that regionally selective alterations in gamma PKC expression are an index of the retrieval of Pavlovian associations formed between a drug and a discrete stimulus, but also that a distinct neural circuitry may underlie Pavlovian stimulus-reward associations in cocaine-experienced rats.

L22 ANSWER 24 OF 39 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:520706 BIOSIS
DOCUMENT NUMBER: PREV200100520706
TITLE: Rat forebrain CaMKII mRNA gene expression in a model of NMDA receptor hypofunction.
AUTHOR(S): De Bartolomeis, A. [Reprint author]; Polese, D. [Reprint author]; de Serpis, A. Amato [Reprint author]; Iasevoli, F. [Reprint author]; D'Urso, G. [Reprint author]; Ambesi-Impiombato, A. [Reprint author]; Muscettola, G. [Reprint author]
CORPORATE SOURCE: Department of Neuroscience and Behavioral Neuroscience, University School of Medicine, Napoli, Italy
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 919. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Nov 2001
Last Updated on STN: 23 Feb 2002

AB Introduction: Cortical and subcortical NMDA receptor hypofunction (NRH) and a subcortical hyperdopaminergy have been implicated in the pathophysiology of schizophrenia. Non competitive NMDA-R antagonists, such as ketamine, may reproduce the glutamate receptor hypofunction in

animal models. The Ca2+/calmoduline-dependent **protein kinase** II (CaMKII), localized in the postsynaptic density region, has been shown to be a signal transduction target in both dopaminergic and glutamatergic neurotransmission and interacts with NMDA-R directly. In this work, we studied CaMKII mRNA gene expression changes in rat forebrain after acute administration of subanaesthetic doses of ketamine. Materials and method: Sprague Dawley male rats were treated, i.p., with ketamine at the dose 12mg/kg, 50mg/kg or with **saline**. CaMKII mRNA was analyzed on thin brain sections by means of quantitative In Situ **Hybridization** Histochemistry (ISHH). Results: Computer assisted analysis of autoradiographic signal has shown in ketamine treated animals a statisticaly significant increase of CaMKII in the following regions: parietal cortex (ANOVA, p=0.0304), frontal cortex (p=0.0122), caudate-putamen (p<0.02), hippocampus (p=0.0103). No statistically significant changes were observed in nucleus accumbens, ventral thalamus and nucleus geniculate. Conclusions: The cortical-subcortical specific increase of CaMKII mRNA, after ketamine administration, suggests a putative involvement of CaMKII in animal models based on NRH.

L22 ANSWER 25 OF 39 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2002414774 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12168769
TITLE: cAMP-dependent protein kinase inhibits proline transport across the rat renal tubular brush border membrane.
AUTHOR: Zelikovic I; Wager-Miller J
CORPORATE SOURCE: Department of Pediatrics, University of Washington School of Medicine, Seattle 98105, USA..
i_zelikovic@rambam.health.gov.il
SOURCE: Bioscience reports, (2001 Oct) Vol. 21, No. 5, pp. 613-26.
Journal code: 8102797. ISSN: 0144-8463.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020810
Last Updated on STN: 20020823
Entered Medline: 20020822

AB Very little is known about the cellular mechanisms controlling renal tubular amino acid transport. cAMP-dependent **protein kinase** (cAK) modulates the activity of several ion channels and pumps in biological membranes. The direct influence of cAK on transmembrane amino acid transport has not been investigated. We studied the effect the cAKmediated phosphorylation on Na+ and Cl(-)-linked proline transport across the rat renal brush border membrane (BBM). cAK bioassay and Western **hybridization** analysis using cAK subunit-specific antibodies demonstrated the presence of the enzyme in the BBM. Brush border membrane vesicles (BBMV) were phosphorylated using the "hyposmotic shock" technique. cAMP, by activating endogenous cAK,and exogenous, highly purified catalytic subunit of cAK inhibited **NaCl**-dependent proline transport by phosphorylated, lysed/resealed BBMV compared with control vesicles. The cAK-mediated inhibition of proline uptake was completely abolished when phosphorylation at the cytoplasmic (inner side) of the membrane was prevented by isosmotic, rather than hyposmotic, phosphorylation. The cAK-induced inhibition of proline

transport was reversed by the specific cAK inhibitor **peptide** , PK1. These data suggest that cAMP-dependent **protein kinase**-mediated phosphorylation modulates Na+(-) and Cl(-)-linked proline transport across the tubular luminal membrane.

L22 ANSWER 26 OF 39 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:390052 SCISEARCH
THE GENUINE ARTICLE: 545XD
TITLE: Random sequencing of cDNAs and identification of mRNAs
AUTHOR: Anderson J V (Reprint); Horvath D P
CORPORATE SOURCE: ARS, USDA, Biosci Res Lab, 1605 Albrecht Blvd, POB 5674, Fargo, ND 58105 USA (Reprint); ARS, USDA, Biosci Res Lab, Fargo, ND 58105 USA
COUNTRY OF AUTHOR: USA
SOURCE: WEED SCIENCE, (SEP-OCT 2001) Vol. 49, No. 5, pp. 590-597.
ISSN: 0043-1745.
PUBLISHER: WEED SCI SOC AMER, 810 EAST 10TH ST, LAWRENCE, KS 66044-8897 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 28
ENTRY DATE: Entered STN: 17 May 2002
Last Updated on STN: 17 May 2002

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB As a first step toward developing a genomics-based research program to study growth and development of underground adventitious shoot buds of leafy spurge, we initiated a leafy spurge expressed sequence tag (EST) database. From the approximately 2,000 clones randomly isolated from a cDNA library made from a population containing growth-induced underground adventitious shoot buds, we have obtained ESTs for 1, 105 cDNAs. Approximately 29% of the leafy spurge EST database consists of expressed genes of unknown identity (hypothetical proteins), and 10% represents ribosomal proteins. The remaining 60% of the database is composed of expressed genes that show BLASTX sequence identity scores of greater than or equal to 80 with known GenBank accessions. Clones showing sequence identity to a Histone H3, a gibberellic acid-responsive gene; Tubulin, and a light-harvesting chlorophyll a/b-binding protein were shown to be differentially expressed in underground adventitious shoot buds of lead, spurge after breaking of dormancy, RNA encoding a putative cyclin-dependent **protein kinase** (CDK)-activating kinase, a gene associated with cell division, and Scarecrow-like 7, a gene involved in GA signaling, were present at similar levels in dormant and growth-induced underground adventitious shoot buds. These data show how even a small EST database can be used to develop a genomics-based research program that will help us identify genes responsive to or involved in the mechanisms controlling underground adventitious shoot bud growth and development.

L22 ANSWER 27 OF 39 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2001225169 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11161828
TITLE: Proline transport in MDCK cells expressing a mutant regulatory subunit of cAMP-dependent protein kinase.
AUTHOR: Zelikovic I; Wager-Miller J
CORPORATE SOURCE: Department of Pediatrics, Division of Nephrology, University of Washington School of Medicine, 4800 Sand

Point Way NW, Seattle, Washington 98105, USA..
i_zelikovic@rambam.health.gov.il
SOURCE: Molecular genetics and metabolism, (2001 Jan) Vol. 72, No. 1, pp. 45-53.
Journal code: 9805456. ISSN: 1096-7192.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010502
Last Updated on STN: 20010502
Entered Medline: 20010426

AB cAMP-dependent **protein kinase** (cAK) regulates the activity of several membrane-bound ion channels and carriers. The role of cAK in regulating the transport of osmoprotective amino acids in the distal tubule is unknown. We examined the regulation of Na(+)- and Cl(-)-dependent proline transport in MDCK cells expressing a mutant murine regulatory subunit (RIalpha(AB)) of cAK. For this purpose, MDCK cells were transfected with an expression vector encoding RIalpha(AB) driven by the metallothionein 1 promoter together with neomycin-resistance (NEO) gene. Stable G418-resistant colonies were isolated that expressed RIalpha(AB) as demonstrated by Northern **hybridization** analysis using a cDNA probe for RIalpha and cAK assay that showed decreased enzyme activity. A clone constitutively expressing high levels of RIalpha(AB) (M(AB)) in a Zn-independent manner and a control clone transfected with the NEO gene alone (M(neo)) were selected for transport studies. We examined the effect of the cAMP-stimulating agents forskolin (F) and IBMX on **NaCl** -dependent uptake of [(3)H]proline by confluent monolayers of transfected MDCK cells. While F/IBMX-induced mean inhibition of proline transport in M(neo) cells was 48 and 45% at 5 and 15 min, respectively, inhibition of proline uptake in M(AB) cells was 9% (5 min) and 0% (15 min). These data demonstrate that the inhibition of **NaCl**-linked proline transport in response to elevated cAMP is reversed in MDCK clones that express mutant cAK and provide evidence that cAK mediates the modulatory action of cAMP on proline transport. cAK may play an important role in controlling transport of proline and other osmoprotective amino acids in the renal tubule.

L22 ANSWER 28 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2000-687353 [67] WPIDS
DOC. NO. NON-CPI: N2000-508150
DOC. NO. CPI: C2000-209242
TITLE: Dual-specificity mitogen activated **protein kinase** phosphatase polypeptides and variants for screening agents which are useful for treating cancer, graft-versus-host disease, autoimmune diseases, and allergies.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): LUCHE, R M; WEI, B
PATENT ASSIGNEE(S): (CEPT-N) CEPTYR INC
COUNTRY COUNT: 92
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000065069	A1	20001102	(200067)*	EN	76

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD
SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000046832	A	20001110 (200109)	
JP 2002542786	W	20021217 (200312)	85
US 6645753	B1	20031111 (200382)	
US 2005075489	A1	20050407 (200525)	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000065069	A1	WO 2000-US11665	20000426
AU 2000046832	A	AU 2000-46832	20000426
JP 2002542786	W	JP 2000-614403	20000426
		WO 2000-US11665	20000426
US 6645753	B1 Provisional	US 1999-131156P	19990427
		US 2000-564357	20000424
US 2005075489	A1 Provisional	US 1999-131156P	19990427
	Cont of	US 2000-564357	20000424
		US 2003-644554	20030819

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000046832	A Based on	WO 2000065069
JP 2002542786	W Based on	WO 2000065069
US 2005075489	A1 Cont of	US 6645753

PRIORITY APPLN. INFO: US 2000-564357 20000424; US
1999-131156P 19990427; US
2003-644554 20030819

AN 2000-687353 [67] WPIDS

AB WO 200065069 A UPAB: 20001223
NOVELTY - An isolated dual-specificity MAP-kinase (DSP-5) or DSP-5 alternate form polypeptide (I) having a fully defined sequence (S) of 340 or 299 amino acids, respectively, or its variant that differs in one or more amino acid deletions, additions, insertions or substitutions at no more than 50% of the residues in (S), such that (I) retains its activity, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (IIa) that encodes at least 10 consecutive amino acids of (I);

(2) an isolated polynucleotide (IIb) that encodes (I);

(3) an expression vector (III) comprising (IIa) or (IIb);

(4) a host cell (IV) transformed or transfected with (III);

(5) an antisense polynucleotide (V) comprising at least 15 consecutive nucleotides complementary to (IIb);

(6) an isolated polynucleotide (VI) that detectably hybridizes to the complement of (IIb) under conditions that include a wash in 0.1X SSC (**saline sodium citrate**) and 0.1% SDS (sodium dodecyl sulfate) at 50 deg. C for 15 minutes;

(7) an expression vector comprising (V) or (VI);

(8) a host cell transformed or transfected with the above

expression vector;

(9) production of (I);

(10) an isolated antibody (VII), or its antigen binding fragment that specifically binds to (I);

(11) a pharmaceutical composition (VIII) comprising (VII) or its fragment;

(12) screening (M1) for an agent that modulates the activity of (I) comprising contacting a candidate agent with (I) or with a cell comprising a DSP-5 promoter or DSP-5 alternate form promoter, operably linked to a polynucleotide encoding a detectable transcript or protein, under conditions and for a time sufficient to permit interactions between (I) or promoter and candidate agent and subsequently evaluating the ability of (I) to dephosphorylate a DSP-5 substrate or DSP-5 alternate form substrate, relative to a predetermined ability of (I) to dephosphorylate the substrate in the absence of candidate agent, or evaluating the expression of the polynucleotide, relative to a predetermined level of expression in the absence of candidate agent, and identifying an agent that modulates the activity of (I);

(13) a DSP-5 substrate or DSP-5 alternate form substrate trapping mutant polypeptide (IX) that differs from (S) in one or more amino acid deletions, additions, insertions or substitutions at no more than 50% of the residues in (S), such that the polypeptide binds to a substrate with an affinity that is not diminished relative to DSP-5 or DSP-5 alternate form, and such that the ability of the polypeptide to dephosphorylate a substrate is reduced relative to DSP-5 or DSP-5 alternate form; and

(14) screening (M2) a molecule for its ability to interact with DSP-5 or DSP-5 alternate form comprising contacting a candidate molecule with (I) under conditions and for a time sufficient to permit the candidate molecule and (I) to interact and detecting the presence or absence of binding of the candidate molecule to (I), and determining whether the candidate molecule interacts with DSP-5 or DSP-5 alternate form.

ACTIVITY - Cytostatic; immunosuppressive; antiallergic.

No biological data is given.

MECHANISM OF ACTION - Modulator of DSP-5 activity.

No biological data is given.

USE - (I) is useful for detecting the expression of DSP-5 or DSP-5 alternate form expression in a biological sample obtained from a patient by contacting the sample with (VII) linked to a support material or detectable marker, or an antigen binding fragment and detecting the level of (VII)/DSP-5 or DSP-5 alternate form complex or by contacting the sample comprising an RNA or cDNA preparation, with (V) or (VI) and detecting in the sample an amount of DSP-5 or DSP-5 alternate form polynucleotide that binds to (V) or (VI), using polymerase chain reaction (PCR) or a hybridization assay. (I) is useful for screening an agent that modulates the activity of DSP-5 or DSP-5 alternate form, which is useful for modulating proliferative response, differentiation or survival of a cell which is present within a patient. The agent modulates a pattern of gene expression, apoptosis or cell cycle and the cell displays contact inhibition of cell growth, anchorage independent growth or altered intracellular adhesion property. The agent is useful for treating a disorder associated with DSP-5 or DSP-5 alternate form activity such as cancer, graft-versus-host disease, autoimmune diseases, allergies, metabolic diseases, abnormal cell growth, abnormal cell proliferation or cell cycle abnormalities.

Dwg.0/6

L22 ANSWER 29 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2000-647239 [62] WPIDS
DOC. NO. NON-CPI: N2000-479666
DOC. NO. CPI: C2000-195798
TITLE: Novel maize Ku80 nucleic acids and their encoded proteins useful for modulating levels of Ku80 in maize plants.
DERWENT CLASS: C06 D16 P13
INVENTOR(S): MAHAJAN, P B
PATENT ASSIGNEE(S): (PION-N) PIONEER HI-BRED INT INC; (MAHA-I) MAHAJAN P B
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000058486	A2	20001005	(200062)*	EN	83
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000036172	A	20001016	(200106)		
EP 1163353	A2	20011219	(200206)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
US 6403860	B1	20020611	(200244)		
JP 2002539838	W	20021126	(200307)		111
US 2003166288	A1	20030904	(200359)		
US 6822077	B2	20041123	(200477)		
AU 778747	B2	20041216	(200508)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2000058486	A2		WO 2000-US5853	20000307
AU 2000036172	A		AU 2000-36172	20000307
EP 1163353	A2		EP 2000-914836	20000307
			WO 2000-US5853	20000307
US 6403860	B1	Provisional	US 1999-126214P	19990325
			US 2000-494810	20000131
JP 2002539838	W		JP 2000-608765	20000307
			WO 2000-US5853	20000307
US 2003166288	A1	Provisional	US 1999-126214P	19990325
		Div ex	US 2000-494810	20000131
			US 2002-160748	20020603
US 6822077	B2	Provisional	US 1999-126214P	19990325
		Div ex	US 2000-494810	20000131
			US 2002-160748	20020603
AU 778747	B2		AU 2000-36172	20000307

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000036172	A	Based on	WO 2000058486

Searcher : Shears 571-272-2528

EP 1163353	A2	Based on	WO 2000058486
JP 2002539838	W	Based on	WO 2000058486
US 2003166288	A1	Div ex	US 6403860
US 6822077	B2	Div ex	US 6403860
AU 778747	B2	Previous Publ.	AU 2000036172
		Based on	WO 2000058486

PRIORITY APPLN. INFO: US 1999-126214P 19990325; US 2000-494810 20000131; US 2002-160748 20020603

AN 2000-647239 [62] WPIDS

AB WO 200058486 A UPAB: 20001130

NOVELTY - Isolated maize Ku80 polynucleotide (PN) (I), (a component of DNA-dependent **Protein Kinase**) comprising a sequence of 2419 base pairs (bp; (I.a)) amplified from a Zea mays nucleic acid library using primers which selectively **hybridize** to loci within (I), encoding a **polypeptide** of 681 amino acids (aa; (I.b)) defined in the specification, is new.

DETAILED DESCRIPTION - Isolated maize Ku80 polynucleotide (PN) (I) comprising:

(a) a sequence with at least 80 % identity with a polynucleotide as in (I.a);

(b) a PN encoding (I.b);

(c) a PN amplified from a Zea mays nucleic acid library using primers which selectively **hybridize** to loci within (I.a);

(d) a PN which selectively **hybridizes** under stringent **hybridization** conditions and a wash in 0.1 multiply SSC (3 mM **sodium chloride**, 3 mM **sodium citrate** and 0.1% sodium dodecyl sulfate (SDS)) at 60 deg. C to (I.a);

(e) a PN as in (I.a);

(f) a PN complementary to (a), (b), (c), (d) or (e); and

(g) a PN comprising at least 30 contiguous nucleotides from a PN as in (a), (b), (c), (d), (e) or (f).

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated maize Ku80 **protein** (II) comprising a sequence of at least 20 contiguous amino acids of , or a sequence with at least 80 % identity to (I.b), encoded by (I);

(2) a recombinant expression cassette (III), comprising (I) operably linked, in sense or anti-sense orientation, to a promoter;

(3) a host cell comprising (III);

(4) a transgenic plant (IV) comprising (III); and

(5) a transgenic seed from (IV).

USE - (I) is useful for modulating the levels of Ku80 in a maize plant by introducing a recombinant expression cassette comprising (I) into the plant cell (claimed). (II) is useful in assays for identifying compounds that bind to and/or increase or decrease the enzymatic activity of (II) and also as immunogen or antigen to produce antibodies which are used in assays for identifying and isolating Ku80 nucleic acids from expression libraries and for identifying homologous **polypeptides** and purifying the **polypeptides**.

Dwg.0/0

L22 ANSWER 30 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2000-195583 [17] WPIDS

CROSS REFERENCE: 2000-195582 [17]; 2000-205722 [18]

DOC. NO. NON-CPI: N2000-144644

DOC. NO. CPI: C2000-060780

TITLE: Novel kappa B-kinase related kinases IKR-1 and IKR-2

used as molecular weight markers and in peptide fragmentation studies.

DERWENT CLASS: B04 D16 S03
INVENTOR(S): BIRD, T A; VIRCA, G D
PATENT ASSIGNEE(S): (IMMV) IMMUNEX CORP
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000008179	A1	20000217	(200017)*	EN	85
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9952527	A	20000228	(200030)		
EP 1019513	A1	20000719	(200036)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2002524047	W	20020806	(200266)		76
US 6605456	B1	20030812	(200355)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2000008179	A1		WO 1999-US17578	19990804
AU 9952527	A		AU 1999-52527	19990804
EP 1019513	A1		EP 1999-937763	19990804
			WO 1999-US17578	19990804
JP 2002524047	W		WO 1999-US17578	19990804
			JP 2000-563802	19990804
US 6605456	B1	Provisional	US 1998-95269P	19980804
		Provisional	US 1998-99973P	19980911
		Provisional	US 1999-118783P	19990205
			WO 1999-US17578	19990804
			US 2000-509800	20000609

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9952527	A	Based on	WO 2000008179
EP 1019513	A1	Based on	WO 2000008179
JP 2002524047	W	Based on	WO 2000008179
US 6605456	B1	Based on	WO 2000008179

PRIORITY APPLN. INFO: US 1999-118783P 19990205; US 1998-95269P 19980804; US 1998-99973P 19980911; US 2000-509800 20000609

AN 2000-195583 [17] WPIDS
CR 2000-195582 [17]; 2000-205722 [18]
AB WO 200008179 A UPAB: 20030828
NOVELTY - Nucleic acids encoding novel kappa B-kinase related kinases, designated IKR-1 and IKR-2, are new.
DETAILED DESCRIPTION - A novel isolated IKR-1 or IKR- 2 nucleic

acid (NA) (I), selected from:

(a) the defined 3385 base pair (bp) or 3219 bp sequences given in the specification;

(b) a NA encoding the defined 717 or 729 amino acid sequences given in the specification;

(c) a NA that **hybridizes** to either strand of a denatured, DNA molecule comprising the sequence of (a) or (b) under moderately stringent conditions (e.g. 50% formamide, 6XSSC (**saline sodium citrate**) at 42 deg. C, and washing in 0.5XSSC and 0.1% SDS (sodium dodecyl sulfate) at 60 deg. C);

(d) a NA derived by in vitro mutagenesis of (a);

(e) a NA degenerate from (a) as a result of the genetic code; and

(f) a NA selected from mouse IKR-1 or IKR-2 DNA, human IKR DNA, allelic variants of mouse or human IKR DNA, and a species homologs of IKR DNA.

INDEPENDENT CLAIMS are also included for the following:

(1) a recombinant vector that directs the expression of (I);

(2) an isolated **polypeptide** (II) encoded by (I), preferably having a molecular weight of 81 or 83 kDa as determined by SDS-PAGE (SDS-polyacrylamide gel electrophoresis) and in a non-glycosylated form, which is especially a **protein kinase**;

(3) isolated (monoclonal) antibodies to (II);

(4) host cell transfected or transduced with the vector of (1);

(5) a method for the production of a IKR-1 or IKR-2 **polypeptide**, comprising culturing the cell of (4) in suitable culture medium under conditions promoting gene expression, and recovering the **polypeptide** from the culture medium (preferably the host cell is selected from bacteria, yeast, plant or animal cells);

(6) a method for determining the molecular weight (MW) of a sample **protein**, comprising comparison of the MW of the sample **proteins** with the MW of (II), wherein the comparison comprises:

(i) application of the sample **protein** and (II) to an acrylamide gel;

(ii) resolution of the sample **protein** and (II) using an electric current; and

(iii) application of a reagent for detecting the sample **protein** and (II);

(7) a kit for detecting the MWs of **peptide** fragments of a sample **protein**, comprising:

(i) a vessel;

(ii) **polypeptide** (II);

(iii) at least one enzyme selected from Asparaginylendopeptidase, Arginylendopeptidase, Achrombobacter protease I, Trypsin, Staphylococcus aureus V8 protease, Endoproteinase Asp-N, and Endoproteinase Lys-C;

(iv) a mutant of (II) derived by in vitro mutagenesis, wherein a site of enzyme cleavage on (II) has been removed; and

(v) fragmented **peptides** derived from (II) by enzymatic cleavage with the selected enzyme, wherein the sample **protein** and (II) are contacted with the same enzyme, and the fragments are detected by the method of (6);

(8) a DAKAR (undefined) **polypeptide** comprising amino acids 1-300 of a defined 717 or 729 amino acid sequence given in the specification;

(9) a method of determining **protein kinase**

activity in which the **protein kinase** moiety is (II) or the **polypeptide** of (8);

(10) a method for identifying substances that affect phosphorylation, comprising:

(i) providing a substance suspected of affecting phosphorylation activity;

(ii) adding (in any order) a **peptide** or **protein** substrate to be phosphorylated, a **polypeptide** kinase consisting of IKR-1 (comprising the 717 amino acid sequence given in the specification), IKR-2 (comprising the 729 amino acid sequence given in the specification), an active domain or fragment thereof, and a source of phosphates;

(iii) incubating for a time and conditions sufficient for kinase-mediated transfer of a phosphate to the substrate;

(iv) measuring the amount of phosphate transferred; and

(v) comparing the amount of phosphate transferred to that transferred in the presence of a standard substance of known phosphorylation-affecting ability;

(11) a method for identifying substances that interfere with the activation of NF-kappaB, comprising:

(i) providing a substance suspected of interfering with the activation of NF-kappaB;

(ii) adding (in any order) peptide or protein substrates to be phosphorylated, a polypeptide kinase consisting of IKR-1 (comprising the 717 amino acid sequence given in the specification), IKR-2 (comprising the 729 amino acid sequence given in the specification), an active domain or fragment thereof, adenosine triphosphate (ATP), a gene with an intact promoter that is activated by NF-kappaB and all other factors necessary for transcription;

(iii) incubating for a time and conditions sufficient for kinase function and transcription of the gene;

(iv) measuring the amount of transcript made;

(v) comparing the amount of transcript made to that made to a standard to determine is the substance interferes with NF- kappaB activation;

(12) a method of designing a molecule that inhibits or enhances the kinase activity of (II), comprising:

(i) determining the three dimensional structure of the polypeptide;

(ii) analyzing this structure for likely binding sites of substrates;

(iii) synthesizing a molecule that incorporates a predictive reactive site; and

(iv) determining the kinase-inhibiting activity or kinase-enhancing activity of the molecule, respectively.

ACTIVITY - Immunomodulatory; antiinflammatory; antimicrobial; cytostatic.

No biological data given.

MECHANISM OF ACTION - The protein function as kinases.

USE - The kappa B-kinase related kinase IKR-1 and IKR- 2 polynucleotides can be used to express the polypeptides, and as probes to identify nucleic acids encoding proteins having kinase activity. IKR-1 and IKR-2 polypeptides and fragmented polypeptides are used for purifying proteins, e.g. to purify binding partner proteins; to measure protein activity, e.g. as quality assurance agents to monitor shelf life and stability of binding partner proteins. They may also be used as research agents, e.g. in assays to determine protein kinase activity, to identify novel molecules involved in signal transduction pathways, and to identify therapeutic compounds, to identify

substances which interfere with the rate of substrate phosphorylation (such compounds would be useful for the treatment of autoimmune, inflammatory, infectious or neoplastic diseases), as molecular weight and isoelectric focusing markers, as controls for peptide fragmentation, identification of unknown proteins, e.g. by comparison with proteins in databases and for preparation of antibodies. The antibodies can be used in assays to detect the presence of the protein, and to purify the protein by immunoaffinity chromatography. The antibodies can also be used to block binding of the IKR polypeptides to their binding partners.

ADVANTAGE - A need exists for polypeptides suitable for use in peptide fragmentation studies, for use in molecular weight measurements, and for use in protein sequencing using tandem mass spectroscopy. This need is met by the present invention.

Dwg.0/4

L22 ANSWER 31 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2000-195582 [17] WPIDS
CROSS REFERENCE: 2000-195583 [17]; 2000-205722 [18]
DOC. NO. NON-CPI: N2000-144643
DOC. NO. CPI: C2000-060779
TITLE: Novel death associated kinase containing ankyrin repeats (DAKAR) used as molecular weight marker and as controls for peptide fragmentation.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BIRD, T A; VIRCA, G D; VIRCA, D G
PATENT ASSIGNEE(S): (IMMV) IMMUNEX CORP
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000008177	A2	20000217	(200017)*	EN	71
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9952527	A	20000228	(200030)		
AU 9954639	A	20000228	(200030)		
EP 1025238	A2	20000809	(200039)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2002526038	W	20020820	(200258)		68
US 6489130	B1	20021203	(200301)		
NZ 503852	A	20030228	(200323)		
US 2003104482	A1	20030605	(200339)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000008177	A2	WO 1999-US17576	19990804
AU 9952527	A	AU 1999-52527	19990804
AU 9954639	A	AU 1999-54639	19990804
EP 1025238	A2	EP 1999-940864	19990804
		WO 1999-US17576	19990804
JP 2002526038	W	WO 1999-US17576	19990804
		JP 2000-563800	19990804

US 6489130	B1	Provisional	US 1998-95269P	19980804
		Provisional	US 1998-99973P	19980911
		Provisional	US 1999-119353P	19990209
			WO 1999-US17576	19990804
			US 2000-509802	20000602
NZ 503852	A		NZ 1999-503852	19990804
			WO 1999-US17576	19990804
US 2003104482	A1	Provisional	US 1998-95269P	19980804
		Provisional	US 1998-99973P	19980911
		Provisional	US 1999-119353P	19990209
		Div ex	WO 1999-US17576	19990804
		Div ex	US 2000-509802	20000602
			US 2002-299327	20021118

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9952527	A	Based on	WO 2000008179
AU 9954639	A	Based on	WO 2000008177
EP 1025238	A2	Based on	WO 2000008177
JP 2002526038	W	Based on	WO 2000008177
US 6489130	B1	Based on	WO 2000008177
NZ 503852	A	Based on	WO 2000008177
US 2003104482	A1	Div ex	US 6489130

PRIORITY APPLN. INFO: US 1999-119353P 19990209; US 1998-95269P 19980804; US 1998-99973P 19980911; US 1999-118783P 19990205; US 2000-509802 20000602; US 2002-299327 20021118

AN 2000-195582 [17] WPIDS

CR 2000-195583 [17]; 2000-205722 [18]

AB WO 200008177 A UPAB: 20030619

NOVELTY - Death associated kinase containing ankyrin repeats (DAKAR) polynucleotides and polypeptides encoded by the polynucleotides are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) an isolated DAKAR nucleic acid (NA) (I) with a sequence selected from:

(a) a defined 2370 base pair sequence given in the specification;

(b) a NA encoding the DAKAR protein sequence of 786 amino acids (A) given in the specification;

(c) a NA that hybridizes to either strand of a denatured, DNA molecule comprising sequence (a) or (b) under moderately stringent conditions;

(d) a NA derived by in vitro mutagenesis of (a);

(e) a NA degenerate from (a) as a result of the genetic code; and

(f) a NA selected from mouse, human, allelic variants of mouse or human, and a species homolog of DAKAR DNA.

(2) a recombinant vector that directs the expression of (I);

(3) an isolated polypeptide (II) encoded by (I),

(4) isolated antibodies to (II);

(5) a host cell transfected or transduced with the vector of (2);

(6) a method for the production of a DAKAR polypeptide, comprising culturing the host cell of (5) in suitable culture medium under conditions promoting gene expression, and recovering the

polypeptide from the culture medium;

(7) a kit for detecting the molecular weights of peptide fragments of a sample protein, comprising a vessel, (II), at least one enzyme which is Asparaginylendopeptidase, Arginylendopeptidase, Achrombobacter protease I, Trypsin, Staphylococcus aureus V8 protease, Endoproteinase Asp-N or Endoproteinase Lys-C, a mutant of (II) derived by in vitro mutagenesis with a site of enzyme cleavage removed and fragmented peptides derived from (II) by enzymatic cleavage with the selected enzyme, where the sample protein and (II) are contacted with the same enzyme, and detected by electrolysis on an acrylamide gel;

(8) a DAKAR polypeptide (III) comprising amino acids 1-785, 17-297 or 471-768 of (A);

(9) an assay of DAKAR **protein kinase** activity where the kinase group is (II), or (III);

(10) a method of inhibiting the kinase activity of DAKAR in a mammal, comprising administering an effective amount of a compound that inhibits the kinase activity of a polypeptide comprising (A), or a fragment of (A);

(11) a method of enhancing the kinase activity of DAKAR in a mammal, comprising administering an effective amount of a compound that enhances the kinase activity of (A), or a fragment of (A); and

(12) a method of designing a molecule that inhibits or enhances the kinase activity of (II), comprising determining the three dimensional structure of the polypeptide, analyzing the structure for likely binding sites of substrates, synthesizing a molecule that incorporates a predictive reactive site and determining the kinase-inhibiting activity or kinase- enhancing activity of the molecule, respectively.

ACTIVITY - No specific activity is given.

MECHANISM OF ACTION - Kinase inhibitor or enhancer.

USE - The death associated kinase containing ankyrin repeats (DAKAR) polynucleotides can be used to express the polypeptides, and as probes to identify nucleic acids encoding proteins having kinase activity. DAKAR polypeptides and fragmented polypeptides are used for purifying proteins, e.g. to measure protein activity; as quality assurance agents to monitor shelf life and stability of binding partner proteins; as research agents, e.g. in assays to determine **protein kinase** activity, to identify novel molecules involved in signal transduction pathways, and to identify therapeutic compounds which may interfere with apoptosis; as molecular weight and isoelectric focusing markers; as controls for peptide fragmentation; identification of unknown proteins, e.g. by comparison with proteins in databases; and for preparation of antibodies.

The antibodies can be used in assays to detect the presence of the protein, and to purify the protein by immunoaffinity chromatography. The antibodies can also be used to block binding of the DAKAR polypeptides to their binding partners.

(II) can be used as a molecular weight marker to determine the molecular weight of a sample protein by electrolysis on an acrylamide gel (claimed).

Compounds that inhibit or enhance the kinase activity of (A), or a fragment of (A) can be used to treat an animal having a disease characterized by overproduction or upregulated production or underproduction or downregulated production of DAKAR (claimed).

Dwg.0/1

L22 ANSWER 32 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2000-136321 [12] WPIDS
CROSS REFERENCE: 2001-450728 [48]; 2002-655433 [70]

DOC. NO. CPI: C2000-041722
TITLE: Nucleic acids encoding a human **protein kinase** homolog useful for preventing, diagnosing and treating cancer, autoimmune/inflammatory disorders and reproductive defects.
DERWENT CLASS: B04 D16
INVENTOR(S): AZIMZAI, Y; BANDMAN, O; CORLEY, N C; GORGONE, G A; GUEGLER, K J; HILLMAN, J L; LU, D A M; TANG, Y T; YUE, H; YANG, Y T
PATENT ASSIGNEE(S): (INCY-N) INCYTE PHARM INC
COUNTRY COUNT: 86
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6013455	A	20000111	(200012)*		38
WO 2000022143	A2	20000420	(200027)	EN	
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 2000013159	A	20000501	(200036)		
EP 1121444	A2	20010808	(200146)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2002527069	W	20020827	(200271)		103

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 6013455	A	US 1998-173581	19981015
WO 2000022143	A2	WO 1999-US24202	19991015
AU 2000013159	A	AU 2000-13159	19991015
EP 1121444	A2	EP 1999-956575	19991015
		WO 1999-US24202	19991015
JP 2002527069	W	WO 1999-US24202	19991015
		JP 2000-576033	19991015

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000013159	A	Based on	WO 2000022143
EP 1121444	A2	Based on	WO 2000022143
JP 2002527069	W	Based on	WO 2000022143

PRIORITY APPLN. INFO: US 1998-173581 19981015
AN 2000-136321 [12] WPIDS
CR 2001-450728 [48]; 2002-655433 [70]
AB US 6013455 A UPAB: 20021105
NOVELTY - Isolated and purified nucleic acids (I) encoding human **protein kinase** homologs (PKHs), are new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:
(i) an isolated and purified polynucleotide (I) encoding a

polypeptide with one of 9 defined sequences given in the specification;

(ii) an isolated and purified polynucleotide (I') that **hybridizes** (in 250 mM **NaCl**, 25 mM **trisodium citrate**, 1% sodium dodecyl sulfate (SDS), 50% formamide and 200 mu g/ml single stranded DNA at 42 deg. C and wash conditions of 15 mM **NaCl**, 1.5 mM **trisodium citrate** and 0.1% SDS at 68 deg. C (these conditions are designated (Y)) to (I);

(iii) a method (II) for detecting a polynucleotide, comprising:

(1) **hybridizing** (I) or (I') to the nucleic acids in the sample to form **hybridization** complexes (under the **hybridization** conditions (Y));

(2) detecting the **hybridization** complexes formed (the presence of **hybridization** complexes correlates with the presence of the polynucleotide in the sample).

ACTIVITY - Cytostatic; antiinflammatory; immunomodulatory; anti-AIDS; antiasthmatic; antidiabetic; antiinfertility.

No biological data given.

MECHANISM OF ACTION - They may be used in gene therapy procedures. (I) or the **protein** may be administered to treat diseases associated with reduced PKH expression and activity by rectifying mutations or deletions in a patient's genome that affect the activity of PKH by expressing inactive and dysfunctional **proteins** or to supplement the patients own production of PKH **polypeptides**.

Vaccine, (I) may also be used for the production of PKH **polypeptides** (both in vivo (as part of a genetic immunization protocol) or in vitro (in a cell culture)), which may then be used as antigens in the production of antibodies specific for PKH which may then be used to down-regulate PKH expression and activity.

USE - (I) and the **protein** it encodes may be used in the prevention, treatment and diagnosis of diseases associated with inappropriate PKH expression such as cancers, autoimmune/inflammatory disorders and reproductive defects. For example, (I) (and vectors containing (I) (Iv)) and the PKH **polypeptide** may be used to treat disorders associated with decreased PKH expression such as cancers (e.g. lymphoma, melanoma and cancers of the breast lung and prostate), autoimmune/inflammatory disorders (e.g. acquired immune deficiency syndrome (AIDS), asthma and diabetes mellitus), disorders of and reproductive defects (e.g. infertility, ovulatory defects and endometriosis and polycystic ovary syndrome).

(I) or (Iv) may be administered to treat diseases by rectifying mutations or deletions in a patient's genome that affect the activity of PKH by expressing inactive **proteins** or to supplement the patients own production of PKH **polypeptides**. Additionally, (I) may be used to produce PKH, according to standard recombinant DNA methodology (for example see Sambrook et al., Molecular Biology: A Laboratory Manual, (1989)), by inserting the nucleic acids into a host cell and culturing the cell to express the **protein** (the **protein** may be expressed either in vitro (in a fermentation culture) or in vivo (as part of a gene therapy procedure)). Conversely, antisense nucleic acid molecules (i.e. (I')) may be administered to down regulate PKH expression by binding with the cells own PKH genes and preventing their expression.

(I) and (I') may also be used as DNA probes in diagnostic assays (e.g. polymerase chain reactions (PCR)) to detect and quantitate the presence of similar nucleic acid sequences in samples, and hence which patients may be in need of restorative therapy (i.e. the method (II)).

They may also be used to study the expression and function of PKH

polypeptides and their role in metabolism.

The PKH **polypeptides** may be used as antigens in the production of antibodies against PKH and in assays to identify modulators (agonists and antagonists) of PKH expression and activity. The anti-PKH antibodies and PKH antagonists may also be used to down regulate PKH expression and activity.

The anti-PKH antibodies may also be used as diagnostic agents for detecting the presence of PKH **polypeptides** in samples (e.g. by enzyme linked immunosorbant assay (ELISA)).

Dwg.0/0

L22 ANSWER 33 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2000-086801 [07] WPIDS
DOC. NO. CPI: C2000-024181
TITLE: Altering the activity of **protein kinase** signaling pathways, used for treating immunosuppressive disorders, e.g. AIDS, proliferative disorders, e.g. cancers or autoimmune diseases.
DERWENT CLASS: B04 D16
INVENTOR(S): ALTMAN, A; HANSSON, V; LEVY, F O; MUNSHI, A; MUSTELIN, T; SKALHEGG, B S; SUNDVOLD, V; TASKEN, K; VANG, T; FINN, O; MUNSH, A; VIDAR, L
PATENT ASSIGNEE(S): (JONE-I) JONES E L; (LAUR-N) LAURAS AS
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9962315	A2	19991202	(200007)*	EN	111
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9941544	A	19991213	(200020)		
EP 1080186	A2	20010307	(200114)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
NO 2000005939	A	20010123	(200118)		
JP 2002516670	W	20020611	(200253)		125
NZ 508359	A	20030725	(200357)		
AU 766674	B	20031023	(200381)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9962315	A2	WO 1999-GB1680	19990527
AU 9941544	A	AU 1999-41544	19990527
EP 1080186	A2	EP 1999-925154	19990527
		WO 1999-GB1680	19990527
NO 2000005939	A	WO 1999-GB1680	19990527
		NO 2000-5939	20001124
JP 2002516670	W	WO 1999-GB1680	19990527
		JP 2000-551593	19990527
NZ 508359	A	NZ 1999-508359	19990527
		WO 1999-GB1680	19990527
AU 766674	B	AU 1999-41544	19990527

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9941544	A	Based on	WO 9962315
EP 1080186	A2	Based on	WO 9962315
JP 2002516670	W	Based on	WO 9962315
NZ 508359	A	Based on	WO 9962315
AU 766674	B	Previous Publ.	AU 9941544
		Based on	WO 9962315

PRIORITY APPLN. INFO: US 1998-114240P 19981230; NO 1998-2419 19980527

AN 2000-086801 [07] WPIDS

AB WO 9962315 A UPAB: 20000209

NOVELTY - A novel method of altering the activity of the **protein kinase** A (PKA) signaling pathway in a cell comprises altering the extent of phosphorylation of one or more PKA substrates, or kinase substrates downstream in the PKA signaling pathway.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a nucleic acid molecule (NAM) comprising a nucleic acid sequence encoding a PKA substrate, or fragment, precursor or functionally equivalent variant, as in (A), where the sequence is modified to alter its susceptibility to phosphorylation by PKA;

(2) a vector comprising a NAM as in (1);

(3) a host cell containing a vector as in (2);

(4) a protein or peptide encoded by a NAM as in (1);

(5) a pharmaceutical composition comprising one or more NAMs, peptides or proteins, encoding or comprising a PKA substrate, downstream kinase substrate, or modified form or fragment, precursor or functionally equivalent variant, or other molecule which alters, preferably inhibits, the phosphorylation of the PKA substrate, or downstream kinase substrate as in (A) or (1)-(4) and one or more excipients and/or diluents.

ACTIVITY - Immunostimulant; cytostatic; anti-HIV; immunosuppressant.

MECHANISM OF ACTION - The novel method alters the activity of the PKA signaling pathway, inhibiting the phosphorylation of downstream kinase substrates.

USE - The pharmaceutical compositions can be used for treating a disorder exhibiting abnormal PKA signaling activity, immunosuppressive disorders or proliferative diseases (claimed). They can be used for treating e.g. HIV infection, AIDS, common variable immunodeficiency or cancers. Conditions in which upregulation of the PKA pathway is required, such as autoimmune disease, e.g. systemic lupus erythematosus, may also be treated.

Dwg.0/15

L22 ANSWER 34 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 1999-403817 [34] WPIDS

CROSS REFERENCE: 1991-237978 [32]; 1992-080013 [10]; 1992-096815 [12]; 1992-096911 [12]; 1992-415799 [50]; 1992-415800 [50]; 1993-018137 [02]; 1993-152175 [18]; 1993-152487 [18]; 1993-227263 [28]; 1993-303152 [38]; 1993-320768 [40]; 1994-026130 [03]; 1994-048786 [06]; 1994-118388 [14]; 1994-135570 [16]; 1994-332803 [41]; 1994-333091 [41]; 1994-333094 [41]; 1994-333100 [41]; 1994-333101 [41];

1995-066911 [09]; 1995-115256 [15]; 1995-115397 [15]; 1995-206893 [27]; 1995-215086 [28]; 1995-246328 [32]; 1995-292881 [38]; 1995-302445 [39]; 1995-402802 [51]; 1996-200879 [20]; 1996-464668 [46]; 1997-011289 [01]; 1997-020468 [02]; 1997-042296 [04]; 1997-042838 [04]; 1997-363002 [33]; 1997-424765 [39]; 1998-008042 [01]; 1998-260961 [23]; 1998-296838 [26]; 1999-024070 [02]; 1999-080503 [07]; 1999-080505 [07]; 1999-120005 [10]; 1999-120932 [10]; 1999-166721 [14]; 1999-214073 [18]; 1999-228583 [19]; 1999-394857 [33]; 1999-404471 [34]; 1999-517980 [43]; 1999-539598 [45]; 1999-561076 [47]; 2000-072074 [06]; 2000-106010 [09]; 2000-160501 [14]; 2000-237346 [20]; 2000-410235 [35]; 2000-586484 [55]; 2000-610851 [58]; 2000-672833 [65]; 2001-025027 [03]; 2001-138117 [14]; 2001-388462 [41]; 2001-407099 [43]; 2001-528597 [58]; 2001-624246 [72]; 2002-054477 [07]; 2002-215022 [27]; 2002-517809 [55]; 2002-519372 [55]; 2002-565044 [60]; 2002-657606 [70]; 2003-438873 [41]; 2003-521529 [49]; 2003-531084 [50]; 2003-566474 [53]; 2003-831271 [77]; 2004-079586 [08]; 2004-106519 [11]; 2004-267636 [25]; 2004-466815 [44]; 2004-561278 [54]; 2004-632911 [61]; 2005-180828 [19]; 2005-214587 [22]; 2005-381495 [39]; 2005-393731 [40]; 2005-532123 [54]

DOC. NO. CPI: C1999-119118
TITLE: New antisense oligonucleotides specific for human protein kinase C useful for diagnosis and treatment of cancer and psoriasis.
DERWENT CLASS: B04 D16
INVENTOR(S): BENNETT, C F; DEAN, N
PATENT ASSIGNEE(S): (ISIS-N) ISIS PHARM INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 5916807	A	19990629	(199934)*		54

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 5916807	A	CIP of	US 1992-852852	19920316
		CIP of	US 1993-89996	19930709
			US 1995-481072	19950607

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 5916807	A	CIP of	US 5703054

PRIORITY APPLN. INFO: US 1995-481072 19950607; US 1992-852852 19920316; US 1993-89996 19930709
AN 1999-403817 [34] WPIDS
CR 1991-237978 [32]; 1992-080013 [10]; 1992-096815 [12]; 1992-096911 [12]; 1992-415799 [50]; 1992-415800 [50]; 1993-018137 [02]; 1993-152175 [18]; 1993-152487 [18]; 1993-227263 [28]; 1993-303152 [38]; 1993-320768 [40]; 1994-026130 [03]; 1994-048786 [06];

Searcher : Shears 571-272-2528

1994-118388 [14]; 1994-135570 [16]; 1994-332803 [41]; 1994-333091 [41]; 1994-333094 [41]; 1994-333100 [41]; 1994-333101 [41]; 1995-066911 [09]; 1995-115256 [15]; 1995-115397 [15]; 1995-206893 [27]; 1995-215086 [28]; 1995-246328 [32]; 1995-292881 [38]; 1995-302445 [39]; 1995-402802 [51]; 1996-200879 [20]; 1996-464668 [46]; 1997-011289 [01]; 1997-020468 [02]; 1997-042296 [04]; 1997-042838 [04]; 1997-363002 [33]; 1997-424765 [39]; 1998-008042 [01]; 1998-260961 [23]; 1998-296838 [26]; 1999-024070 [02]; 1999-080503 [07]; 1999-080505 [07]; 1999-120005 [10]; 1999-120932 [10]; 1999-166721 [14]; 1999-214073 [18]; 1999-228583 [19]; 1999-394857 [33]; 1999-404471 [34]; 1999-517980 [43]; 1999-539598 [45]; 1999-561076 [47]; 2000-072074 [06]; 2000-106010 [09]; 2000-160501 [14]; 2000-237346 [20]; 2000-410235 [35]; 2000-586484 [55]; 2000-610851 [58]; 2000-672833 [65]; 2001-025027 [03]; 2001-138117 [14]; 2001-388462 [41]; 2001-407099 [43]; 2001-528597 [58]; 2001-624246 [72]; 2002-054477 [07]; 2002-215022 [27]; 2002-517809 [55]; 2002-519372 [55]; 2002-565044 [60]; 2002-657606 [70]; 2003-438873 [41]; 2003-521529 [49]; 2003-531084 [50]; 2003-566474 [53]; 2003-831271 [77]; 2004-079586 [08]; 2004-106519 [11]; 2004-267636 [25]; 2004-466815 [44]; 2004-561278 [54]; 2004-632911 [61]; 2005-180828 [19]; 2005-214587 [22]; 2005-381495 [39]; 2005-393731 [40]; 2005-532123 [54]

AB US 5916807 A UPAB: 20051216

NOVELTY - A method of inhibiting the expression of human **protein kinase** C (PKC) in cells comprises contacting the cells with an antisense oligonucleotide which has up to 50 nucleotide units and comprises a nucleotide sequence of 17-20 bp given in the specification.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a method of inhibiting the expression of human **protein kinase** C- alpha comprising contacting cells with an antisense oligonucleotide which has up to 50 nucleotide units and comprises nucleotide sequence (I), (II) which is free of a 5'-dimethoxytrityl moiety, or (III).

5'-GTTCTCGCTGGTGAGTTTCA-3' (I)

5'-AAAACGTCAGCCATGGTCCC-3' (II)

5'-GAGACCCTGAACAGTTGATC-3' (III)

ACTIVITY - Cytostatic; antipsoriatic.

MDA-MB231 human breast carcinoma cells were transplanted into nude mice to establish MDA-MB231 tumors. After two weeks **protein kinase** C- alpha (PKC- alpha) oligonucleotides with sequence 5'-GTTCTCGCTGGTGAGTTTCA-3' (I) and 5'-AAAACGTCAGCCATGGTCCC-3' (II) were administered intravenously in **saline** daily for 14 days at 60 and 6 mg/kg. A control oligonucleotide without significant sequence homology to the PKC mRNA target and a **saline** control were also administered at the same doses. The growth of the tumor was monitored for the 2 week administration period.

Both PKC- alpha oligonucleotides were found to significantly inhibit the tumor growth at both dosage rates. The relative tumor size at the start of treatment was 1 and after the 14 days had risen to 7.5 with the controls and 2.8 with (I) administered at 60 mg/kg and 3.75 with (I) administered at 6 mg/kg. Administration of (II) inhibited growth of the tumor to the relative size of 2.2 at 60 mg/kg and 3.5 at 6 mg/kg.

MECHANISM OF ACTION - **Protein kinase** C inhibitor.

USE - The antisense oligonucleotides modulate **hybridize** to messenger RNA from the PKC gene which results in modulation of

expression of the PKC gene. This means they can be used for diagnosis, therapeutic or prophylactic treatment of PKC associated diseases such as cancer and psoriasis, and as research agents. Abnormal proliferative states in tissue from patients suspected of having a hyperproliferative disease e.g. cancer, psoriasis can be diagnosed. Tumors associated with PKC can be distinguished from tumors which are not PKC associated to allow an efficacious treatment regime to be used.

ADVANTAGE - The antisense oligonucleotides have specific activity so are able to modulate PKC activity without producing side effects and with greater effectiveness than observed from administration of current agents.

Dwg.0/15

L22 ANSWER 35 OF 39 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 1997-087168 [08] WPIDS

DOC. NO. NON-CPI: N1997-071805

DOC. NO. CPI: C1997-028325

TITLE: Interleukin-1 receptor-associated **protein kinase** and related DNA - used to develop prods. for the diagnosis and treatment of e.g. infection, genetic diseases, neoplasia, inflammation or hypersensitivity.

DERWENT CLASS: B04 D16 S03

INVENTOR(S): CAO, Z; CROSTON, G E; GOEDDEL, D V

PATENT ASSIGNEE(S): (TULA-N) TULARIK INC

COUNTRY COUNT: 22

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9700690	A1	19970109	(199708)*	EN	33
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9661766	A	19970122	(199719)		
US 5654397	A	19970805	(199737)		11
EP 839045	A1	19980506	(199822)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
AU 702844	B	19990304	(199921)		
JP 11509085	W	19990817	(199943)		32
CA 2225450	C	20010529	(200134)	EN	

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9700690	A1		WO 1996-US9193	19960605
AU 9661766	A		AU 1996-61766	19960605
US 5654397	A	Cont of	US 1995-494006	19950623
			US 1996-587889	19960116
EP 839045	A1		EP 1996-919140	19960605
			WO 1996-US9193	19960605
AU 702844	B		AU 1996-61766	19960605
JP 11509085	W		WO 1996-US9193	19960605
			JP 1997-503857	19960605
CA 2225450	C		CA 1996-2225450	19960605
			WO 1996-US9193	19960605

FILING DETAILS:

Searcher : Shears 571-272-2528

PATENT NO	KIND		PATENT NO
AU 9661766	A	Based on	WO 9700690
EP 839045	A1	Based on	WO 9700690
AU 702844	B	Previous Publ.	AU 9661766
		Based on	WO 9700690
JP 11509085	W	Based on	WO 9700690
CA 2225450	C	Based on	WO 9700690

PRIORITY APPLN. INFO: US 1995-494006 19950623; US 1996-587889 19960116

AN 1997-087168 [08] WPIDS

AB WO 9700690 A UPAB: 19970220

An isolated human Interleukin-1 Receptor-Associated **Protein Kinase** (IRAK) which migrates under SDS-PAGE at an apparent mol. weight of approx. 100 kD is claimed. Also claimed are: (1) an isolated human IRAK comprising a kinase domain (A) having a 712 amino acid sequence (given in the specification), residues 212-523; (2) an isolated nucleic acid (I) encoding (A); and (3) an isolated first nucleic acid comprising a 3590 bp sequence (given in the specification) or capable of specifically **hybridising** with the sequence and remaining bound at a reduced final wash stringency of 20% formamide in 0.9M **saline**/0.09M **sodium citrate** (SSC) buffer at a temperature of 42deg.C.

USE - The prods. can be used for identifying lead cpds. for agents useful in the diagnosis or treatment of disease associated with IRAK activity (claimed). Target indications include infection, genetic disease, cell growth and regulatory dysfunction, such as neoplasia, inflammation, and hypersensitivity.

Dwg.0/0

ABEQ US 5654397 A UPAB: 19970915

Isolated human interleukin-1 receptor-associated **protein kinase** (IRAK) polypeptides comprising amino acids 1-120 [self-association domain] and/or amino acids 212-523 [active kinase domain] of a sequence of 712 amino acids given in the specification are new. Also claimed is an isolated nucleic acid encoding the kinase domain.

USE - The human IRAK sequences can be used to screen for (a) compounds useful for the diagnosis or treatment of diseases associated with interleukin-1 (IL-1) signal transduction (especially inflammation) and (b) compounds useful for the diagnosis or treatment of diseases associated with IRAK activity.

Dwg.0/0

L22 ANSWER 36 OF 39 MEDLINE on STN DUPLICATE 5

ACCESSION NUMBER: 96301332 MEDLINE

DOCUMENT NUMBER: PubMed ID: 8722631

TITLE: Regulation of steady-state concentrations of messenger ribonucleic acid encoding prostaglandin F2 alpha receptor in ovine corpus luteum.

AUTHOR: Juengel J L; Wiltbank M C; Meberg B M; Niswender G D

CORPORATE SOURCE: Animal reproduction and biotechnology Laboratory, Colorado State University, Fort Collins 80523-1683, USA.

CONTRACT NUMBER: HD11590 (NICHD)

SOURCE: Biology of reproduction, (1996 May) Vol. 54, No. 5, pp. 1096-102.
Journal code: 0207224. ISSN: 0006-3363.

Searcher : Shears 571-272-2528

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19961008
Last Updated on STN: 19961008
Entered Medline: 19960926

AB To investigate the regulation of ovine luteal receptors for prostaglandin F2 alpha (PGF2 alpha), reverse transcription-polymerase chain reaction was used to produce a 284-bp partial cDNA that was 98% identical to that reported for the bovine PGF2 alpha receptor (PGF2 alpha-R). In situ **hybridization** localized mRNA for PGF2 alpha-R specifically to large luteal cells. In experiment 1, pools of luteal tissue (n = 4/day) collected from ewes on Days 3, 6, 9, 12, and 15 of the estrous cycle were analyzed for mRNA encoding PGF2 alpha-R. There was no difference in mean steady-state concentrations of mRNA encoding PGF2 alpha-R among any of the days studied (range = 2.3 +/- 0.3 to 3.5 +/- 0.7 fmol PGF2 alpha-R mRNA/ microgram poly[A]+ RNA as assessed by slot-blot **hybridization**). In experiment 2, ewes on Day 11 or Day 12 of the estrous cycle were administered PGF2 alpha, and corpora lutea were collected 4, 12, or 24 h later (n = 4-5 per time point). Nontreated (n = 4) or **saline**-treated (n = 4) ewes served as controls. Luteal concentrations of mRNA encoding PGF2 alpha-R were decreased ($p < 0.05$) at 4, 12, and 24 h after injection of PGF2 alpha. In experiment 3, ewes (midluteal phase) were administered **saline**, PGF2 alpha, phorbol 12-myristate 13-acetate (PMA), or LH via ovarian arterial injection, and luteal tissue was collected 0, 4, 12, or 24 h later (n = 3-4 per treatment per time). Steady-state concentrations of mRNA encoding PGF2 alpha-R were decreased ($p < 0.05$) by PGF2 alpha and PMA treatment (4 and 12 h) but were increased ($p < 0.05$) at 24 h after LH treatment. In summary, 1) mRNA encoding PGF2 alpha-R was localized to large luteal cells; 2) concentrations of mRNA encoding PGF2 alpha-R did not vary during the estrous cycle; 3) treatment with PGF2 alpha or PMA to activate **protein kinase** C decreased concentrations of PGF2 alpha-R mRNA within 4 h of treatment; and 4) administration LH increased concentrations of mRNA encoding PGF2 alpha-R 24 h following injection.

L22 ANSWER 37 OF 39 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 95093475 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8000427
TITLE: A NaCl-regulated plant gene encoding a brain protein homology that activates ADP ribosyltransferase and inhibits protein kinase C.
AUTHOR: Chen Z; Fu H; Liu D; Chang P F; Narasimhan M; Ferl R; Hasegawa P M; Bressan R A
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA 02111.
SOURCE: The Plant journal : for cell and molecular biology, (1994 Nov) Vol. 6, No. 5, pp. 729-40.
Journal code: 9207397. ISSN: 0960-7412.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-S76737
ENTRY MONTH: 199501

Searcher : Shears 571-272-2528

ENTRY DATE: Entered STN: 19950215
Last Updated on STN: 19980206
Entered Medline: 19950125

AB A cDNA clone pCZ1, with a 1.1 kb insert, was isolated from a **NaCl**-adapted tobacco cell cDNA library that encodes an apparently full-length 29 kDa **protein** (251 amino acids) with a calculated pI of 5.7. The encoded **peptide** had a high amino acid sequence identity with bovine 14-3-3 **protein** which was originally found as an abundant **protein** in the animal central nervous system. Recently, **proteins** with sequence identity to 14-3-3 **protein** have also been found in plants, insects and yeast, and appear to have diverse physiological functions. Similar to the bovine brain 14-3-3 **protein**, the recombinant pCZ1 **protein** stimulated ADP-ribosylation of **protein** substrate by ADP-ribosyltransferase from the plant and animal pathogenic bacterium Pseudomonas aeruginosa. This recombinant **protein** also inhibited **protein kinase** C activity in vitro. Southern blot analyses indicated that most likely five genes encoding 14-3-3-like **proteins** are present in tobacco. The pCZ1 cDNA insert **hybridized** to a single mRNA of 1.1 kb from cultured tobacco cells. The level of this mRNA transcript in tobacco cells was downregulated upon adaptation to **NaCl** but was unaffected by short-term treatment with **NaCl**, ABA or ethylene. In tobacco plants, expression of transcript that **hybridized** to pCZ1 was tissue specific, and was most abundant in roots and flower parts. Monoclonal antibody raised against GF14 **protein**, a maize **protein** with substantial sequence identity with 14-3-3 **protein** detected two bands on SDS-PAGE of total **proteins** from unadapted tobacco cells and only a single band from cells adapted to **NaCl**. The GF14 antibody was also used to illustrate that the G-box element of a salt-induced gene is associated with a 14-3-3-type **protein**.

L22 ANSWER 38 OF 39 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 92250540 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1374392
TITLE: Cloning and sequencing of a rabbit cDNA encoding an intestinal and kidney-specific Na+/H+ exchanger isoform (NHE-3).
AUTHOR: Tse C M; Brant S R; Walker M S; Pouyssegur J; Donowitz M
CORPORATE SOURCE: Department of Physiology, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205.
CONTRACT NUMBER: R01DK26523 (NIDDK)
R29DK43778 (NIDDK)
T32DK07632 (NIDDK)
+
SOURCE: The Journal of biological chemistry, (1992 May 5) Vol. 267, No. 13, pp. 9340-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M74319; GENBANK-M74320; GENBANK-M74321; GENBANK-M74322; GENBANK-M74323; GENBANK-M74324; GENBANK-M76378; GENBANK-M85300; GENBANK-M85301; GENBANK-M87007

Searcher : Shears 571-272-2528

ENTRY MONTH: 199206
ENTRY DATE: Entered STN: 19920619
Last Updated on STN: 19960129
Entered Medline: 19920605

AB We previously cloned, sequenced, and expressed two distinct mammalian Na+/H+ exchanger isoforms (NHE-1 and NHE-2). We report here the cloning of a composite cDNA which encodes a third mammalian isoform (NHE-3), which is expressed specifically in intestine and kidney. The **protein** deduced from the longest open reading frame of this composite sequence has 832 amino acids with a calculated Mr of 92,747. The hydrophobicity plot of NHE-3 is very similar to that of NHE-1 and NHE-2. NHE-3 is also predicted to have 10-12 membrane-spanning domains and a long cytoplasmic domain which contains putative **protein kinase** phosphorylation motifs. NHE-3 exhibits overall 41% amino acid identity with NHE-1. NHE-3 is likely a glycoprotein as it has one potential N-linked glycosylation site, which is conserved in all NHEs identified. Northern blot analysis of poly(A+) RNA isolated from rabbit ileum using NHE-3 cDNA as a probe **hybridized** to a single 5.4-kilobase transcript. More detailed tissue distribution of message was performed by ribonuclease protection assay. It was found that NHE-3 message is only expressed in intestine and kidney, with the kidney cortex having the most abundant message, followed by intestine and kidney medulla. In intestine, ileum and ascending colon have the same amount of message, with much lesser amounts in jejunum. The message is absent from duodenum and descending colon, which lack the neutral **NaCl** absorptive process. Thus, NHE-3 might be involved in Na+ absorption in intestinal and renal epithelial cells.

L22 ANSWER 39 OF 39 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 83257320 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6191775
TITLE: Characterization of a messenger RNA transport protein.
AUTHOR: Moffett R B; Webb T E
CONTRACT NUMBER: GM/CA 29221 (NIGMS)
P-30-CA-16058-09 (NCI)
SOURCE: Biochimica et biophysica acta, (1983 Aug 2) Vol. 740, No. 3, pp. 231-42.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198309
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19980206
Entered Medline: 19830909

AB A cytoplasmic **protein** which facilitates the energy-dependent transport of mRNA from isolated nuclei to a specified medium has been further characterized, since it could have relevance to the mechanism of mRNA nucleo-cytoplasmic transport in vivo. This **protein** is now shown, by cDNA **hybridization** analysis using appropriate recombinant probes, to be obligatory for the transport of alpha 2u-globulin and albumin mRNA from male rat liver nuclei. It is concentrated in the cytoplasm. When isolated under conditions where they retain nuclear **proteins**, the nuclei contain less than 2% of the total mRNA transport activity. Approx. 20% is recovered in the cytosol, while the rest (80%) copurifies with the messenger ribonucleoproteins in the polyribosome fraction. The **protein**

is eluted from the poly A-messenger ribonucleoproteins between 0.25 and 0.50 M **NaCl**. The activities of the cytosolic- and messenger ribonucleoprotein-derived transport **proteins** were mutually additive below saturation of the transport system. Further, the activities of both fractions were increased when they were fortified with the catalytic subunit of the cAMP-dependent **protein kinase** in the presence of ATP. On the other hand, **protein kinase**-induced thiophosphorylation of the **protein** with ATP[S] decreased transport activity. The molecular weight of the transport **protein** from either cell compartment as judged by molecular sieving is approx. 35,000. It has now been purified 2000-fold and requires manganese ions and serum albumin for stabilization of activity. The highly purified transport factor from the cytosol is tentatively assigned a molecular weight of 32,000 by SDS-polyacrylamide gel electrophoresis.

(FILE 'USPATFULL' ENTERED AT 14:11:01 ON 14 MAR 2006)

L4	445	SEA FILE=REGISTRY ABB=ON PLU=ON SODIUM CHLORIDE ?/CN
L5	5	SEA FILE=REGISTRY ABB=ON PLU=ON ("SODIUM CITRATE"/CN OR "SODIUM CITRATE (NA2O7C6H6)"/CN OR "SODIUM CITRATE (NA3C6D5O7)"/CN OR "SODIUM CITRATE (NA3C6H5O7)"/CN OR "SODIUM CITRATE (NAC6H7O7)"/CN)
L7	1698	SEA FILE=REGISTRY ABB=ON PLU=ON PROTEIN KINASE ?/CN
L8	165813	SEA FILE=CAPLUS ABB=ON PLU=ON L7 OR PROTEIN KINASE
L9	430653	SEA FILE=CAPLUS ABB=ON PLU=ON L4 OR (NA OR SODIUM)(W)(CL OR CHLORIDE) OR NACL OR SALINE
L10	20942	SEA FILE=CAPLUS ABB=ON PLU=ON L5 OR (NA OR TRISODIUM OR SODIUM)(W)CITRATE OR CITRA
L26	527	SEA FILE=USPATFULL ABB=ON PLU=ON L8(S)L9
L27	23	SEA FILE=USPATFULL ABB=ON PLU=ON L26(S)L10
L28	21	SEA FILE=USPATFULL ABB=ON PLU=ON L27(S)(HYBRIDIS? OR HYBRIDIZ?)

L29 21 S L28 NOT L3

L29 ANSWER 1 OF 21 USPATFULL on STN

ACCESSION NUMBER:	2005:299813 USPATFULL
TITLE:	Regulation of human serine-threonine protein kinase
INVENTOR(S):	Kohler, Rainer H., Beverly, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005261482	A1	20051124	
APPLICATION INFO.:	US 2003-451375	A1	20011227	(10)
	WO 2001-EP15320		20011227	
			20031118	PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-259215P	20010103 (60)
	US 2003-306468P	20010720 (60)
	US 2003-308098P	20010730 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001, US
NUMBER OF CLAIMS:	42
EXEMPLARY CLAIM:	1-71

NUMBER OF DRAWINGS: 24 Drawing Page(s)
LINE COUNT: 3304
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents that regulate human serine-threonine protein kinase and reagents which bind to human serine-threonine protein kinase gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, diabetes, COPD, and peripheral and central nervous system disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 2 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2005:81430 USPATFULL
TITLE: Mutated aqp, method for detecting cancer using the same, dna chip having oligonucleotides of said mutated aqp sequence
INVENTOR(S): Moon, Woo-Chul, Seoul, KOREA, REPUBLIC OF
Moon, Chul-so, Houston, TX, UNITED STATES
Moon, Young-ho, Kwangmyung, KOREA, REPUBLIC OF
Kim, Byung-gu, Seoul, KOREA, REPUBLIC OF
Kim, Dong-hwan, Kwangmyung, KOREA, REPUBLIC OF
Shin, Chan-jae, Seoul, KOREA, REPUBLIC OF
Um, Tae-han, Seoul, KOREA, REPUBLIC OF
Kim, Hwa-su, Seoul, KOREA, REPUBLIC OF
Song, Mi-kyung, Seoul, KOREA, REPUBLIC OF
Kim, Hyeung-jae, Kyungsangnamdo, KOREA, REPUBLIC OF
Song, Seok-beom, Daejeon, KOREA, REPUBLIC OF

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005069872	A1	20050331	
APPLICATION INFO.:	US 2004-363925	A1	20040105	(10)
	WO 2001-KR1528		20010910	

	NUMBER	DATE
PRIORITY INFORMATION:	KR 2000-53821	20000909

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 76 Drawing Page(s)
LINE COUNT: 1543
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to mutation genes of the AQP(aquaporin), a method for detecting cancer using mutations and expressions of the AQP and a DNA chip possessing oligonucleotides of mutated AQP base sequence. In case of the present method for detecting cancer and DNA chip using the AQP's mutations and expressions, it is highly accurate, rapid and effective in cancer diagnosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 3 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2005:62902 USPATFULL
TITLE: Regulation of human serine/threonine protein kinase

INVENTOR(S): Kohler, Rainer H., Hamilton, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2005053938	A1	20050310	
APPLICATION INFO.:	US 2004-493211	A1	20041022	(10)
	WO 2002-EP13268		20021126	

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-333131P	20011127	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: CLM-01-17
NUMBER OF DRAWINGS: 26 Drawing Page(s)
LINE COUNT: 5069
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Reagents that regulate human serine/threonine protein kinase and reagents which bind to human serine/threonine protein kinase gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, diabetes, CNS disorders, respiratory disorders, COPD, cardiovascular disorders, dermatological disorders, gastrointestinal or liver diseases, hematological disorders, muscle-skeletal disorders, reproduction disorders, or urological disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 4 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:321016 USPATFULL
TITLE: Regulation of human dcamkl1-like serine/threonine protein kinase
INVENTOR(S): Xiao, Yonghong, Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004253669	A1	20041216	
APPLICATION INFO.:	US 2004-487090	A1	20040805	(10)
	WO 2002-EP9282		20020820	

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-313809P	20010822	(60)
	US 2002-378413P	20020508	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: CLM-01-17
NUMBER OF DRAWINGS: 28 Drawing Page(s)
LINE COUNT: 4061
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Reagents that regulate human DCAMKL1-like serine/threonine protein kinase and reagents which bind to human DCAMKL1-like serine/threonine protein kinase gene products can play a role in

preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, diabetes, CNS disorders, COPD, asthma or cardiovascular disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 5 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:307125 USPATFULL
TITLE: Regulation of human nek-like serine/threonine protein kinase
INVENTOR(S): Xiao, Yonghong, Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004241796	A1	20041202	
APPLICATION INFO.:	US 2004-481455	A1	20040526	(10)
	WO 2002-EP6948		20020624	

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-300068P	20010625 (60)
	US 2001-336704P	20011207 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: CLM-01-17
NUMBER OF DRAWINGS: 33 Drawing Page(s)
LINE COUNT: 3750
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents that regulate human NEK-like serine/threonine protein kinase and reagents which bind to human NEK-like serine/threonine protein kinase gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, particularly colon cancer, cardiovascular disorders, CNS disorders, COPD or diabetes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 6 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:292223 USPATFULL
TITLE: Guanylate-binding protein
INVENTOR(S): Pennica, Diane, Burlingame, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc. (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004229307	A1	20041118	
APPLICATION INFO.:	US 2003-659549	A1	20030910	(10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-643657, filed on 17 Aug 2000, GRANTED, Pat. No. US 6642024 Continuation of Ser. No. US 1998-15089, filed on 29 Jan 1998, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Attention of William J. Wood, Gates & Cooper LLP, Howard Hughes Center, 6701 Center Drive West, Suite 1050, Los Angeles, CA, 90045

NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: CLM-001-9
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 3952
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A member of the guanylate-binding protein family, designated GBP-4, is provided. Also provided are isolated nucleic acid encoding GBP-4, vectors and host cells containing such nucleic acid molecule, and a method for producing the GBP-4 recombinantly.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 7 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:221768 USPATFULL
TITLE: Regulation of human protein kinase-like protein
INVENTOR(S): Smolyar, Alex, Brookline, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004171539	A1	20040902	
APPLICATION INFO.:	US 2004-471762	A1	20040413	(10)
	WO 2002-EP2887		20020315	

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-276055P	20010316 (60)
	US 2001-324053P	20010924 (60)
	US 2001-326458P	20011003 (60)
	US 2001-337124P	20011210 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 71
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 3894
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents that regulate human protein kinase-like protein and reagents which bind to human protein kinase-like gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, CNS disorders, COPD, obesity, diabetes, and cardiovascular disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 8 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:178426 USPATFULL
TITLE: Regulation of human serine/threonine protein kinase-like protein
INVENTOR(S): Kohler, Rainer H., Beverly, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004137593	A1	20040715	
APPLICATION INFO.:	US 2003-473144	A1	20031008	(10)
	WO 2002-EP4080		20020412	

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 70
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 24 Drawing Page(s)
LINE COUNT: 3957
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents that regulate human serine/threonine protein kinase-like protein and reagents which bind to human serine/threonine protein kinase-like gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, CNS disorders, COPD, cancer, and diabetes, obesity and urology disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 9 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:132993 USPATFULL
TITLE: REGULATION OF HUMAN SERINE-THREONINE PROTEIN KINASE
INVENTOR(S): Smolyar, Alex, Brookline, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004101529	A1	20040527	
APPLICATION INFO.:	US 2003-399136	A1	20030414	(10)
	WO 2001-EP11893		20011015	

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 71
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 2805
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents which regulate human serine-threonine protein kinase and reagents which bind to human serine-threonine protein kinase gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, CNS disorders, diabetes, asthma, and COPD.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 10 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:101187 USPATFULL
TITLE: Regulation of human wee1-like serine/threonine protein kinase
INVENTOR(S): Kohler, Rainer H., Beverly, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004077049	A1	20040422	
APPLICATION INFO.:	US 2003-470726	A1	20030731	(10)
	WO 2002-EP912		20020130	

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 71

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3930
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents that regulate human WEE1-like serine/threonine protein kinase and reagents which bind to human WEE1-like serine/threonine protein kinase gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, peripheral and central nervous system disorders, genitourinary disorders, cardiovascular disorders, and COPD.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 11 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2004:57373 USPATFULL
TITLE: Regulation of human serine-threonine protein kinase
INVENTOR(S): Zhu, Zhimin, Weban, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004043375	A1	20040304	
APPLICATION INFO.:	US 2003-398920	A1	20030710	(10)
	WO 2001-EP11925		20011016	

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 71
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 3965
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents which regulate human serine-threonine protein kinase and reagents which bind to human serine-threonine protein kinase gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cancer, CNS disorders, diabetes, and COPD.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 12 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2003:152829 USPATFULL
TITLE: Sperm-specific AKAP protein genes and uses
INVENTOR(S): Carr, Daniel W., Portland, OR, UNITED STATES
Vijayaraghavan, Srinivasan, Kent, OH, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003104495	A1	20030605	
APPLICATION INFO.:	US 2002-244488	A1	20020916	(10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-268480, filed on 16 Mar 1999, GRANTED, Pat. No. US 6451528
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 2251
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides isolated nucleic acid encoding sperm-specific protein kinase A anchoring proteins from mammalian species, recombinant expression constructs encoding said mammalian sperm-specific protein kinase A anchoring proteins, cells transformed with said constructs, and homogenous preparations of these proteins prepared using recombinant genetic techniques. The invention also provides analytic tools such as polyclonal antisera and monoclonal antibodies specific for said sperm-specific protein kinase A anchoring proteins of the invention. The invention also provides methods for isolating nucleic acid encoding mammalian sperm-specific protein kinase A anchoring proteins and recombinant genetic methods for producing said mammalian sperm-specific protein kinase A anchoring proteins of the invention. The invention particularly provides high throughput screening methods for screening compounds that can disrupt binding between protein kinase A and the sperm-specific protein kinase A anchoring protein and methods for determining and producing compounds, particularly antisense oligonucleotides, for inhibiting or preventing expression of sperm-specific protein kinase A anchoring protein in sperm, spermatids, or progenitor cells thereof. These methods are useful for development of effective, male-specific contraceptives, which are also provided by the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 13 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2003:86312 USPATFULL
TITLE: Regulation of human serine/threonine protein kinase
INVENTOR(S): Smolyar, Alex, Brookline, MA, UNITED STATES
Horner, Emma Jane, Berkshire, UNITED KINGDOM
Thelwell, Craig, Slough, UNITED KINGDOM
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Leverkusen, GERMANY, FEDERAL REPUBLIC OF (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003059918	A1	20030327	
APPLICATION INFO.:	US 2002-162706	A1	20020606	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-348601P	20020117	(60)
	US 2001-330578P	20011025	(60)
	US 2001-323100P	20010919	(60)
	US 2001-296164P	20010607	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 52
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 3771
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents that regulate a novel human serine/threonine protein kinase and reagents which bind to the human serine/threonine protein kinase

gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, obesity, genitourinary disorders, CNS disorders, diabetes, cancer, and COPD.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 14 OF 21 USPATFULL on STN
ACCESSION NUMBER: 2002:238817 USPATFULL
TITLE: Sperm-specific AKAP protein genes and uses
INVENTOR(S): Carr, Daniel W., Portland, OR, United States
Vijayaraghavan, Srinivasan, Kent, OH, United States
PATENT ASSIGNEE(S): Oregon Health & Sciences University, Portland, OR, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6451528	B1	20020917
APPLICATION INFO.:	US 1999-268480		19990316 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Saidha, Tekchand
ASSISTANT EXAMINER: Kerr, Kathleen
LEGAL REPRESENTATIVE: McDonnell Boehnen Hulbert & Berghoff
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT: 2243
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides isolated nucleic acid encoding sperm-specific protein kinase A anchoring proteins from mammalian species, recombinant expression constructs encoding said mammalian sperm-specific protein kinase A anchoring proteins, cells transformed with said constructs, and homogenous preparations of these proteins prepared using recombinant genetic techniques. The invention also provides analytic tools such as polyclonal antisera and monoclonal antibodies specific for said sperm-specific protein kinase A anchoring proteins of the invention. The invention also provides methods for isolating nucleic acid encoding mammalian sperm-specific protein kinase A anchoring proteins and recombinant genetic methods for producing said mammalian sperm-specific protein kinase A anchoring proteins of the invention. The invention particularly provides high throughput screening methods for screening compounds that can disrupt binding between protein kinase A and the sperm-specific protein kinase A anchoring protein and methods for determining and producing compounds, particularly antisense oligonucleotides, for inhibiting or preventing expression of sperm-specific protein kinase A anchoring protein in sperm, spermatids, or progenitor cells thereof. These methods are useful for development of effective, male-specific contraceptives, which are also provided by the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 15 OF 21 USPATFULL on STN
ACCESSION NUMBER: 1998:108249 USPATFULL
TITLE: Prostaglandin E receptors, their DNA and production
INVENTOR(S): Ichikawa, Atsushi, Besshohonmachi, Japan
Narumiya, Shuh, Kyoto, Japan

PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5804415		19980908
APPLICATION INFO.:	US 1996-685945		19960722 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-390162, filed on 17 Feb 1995, now patented, Pat. No. US 5576192 which is a continuation of Ser. No. US 1993-24179, filed on 23 Feb 1993, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1992-36580	19920224
	JP 1992-64889	19920323

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Teng, Sally P.
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 1001
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are (1) a protein capable of receiving PGE, (2) a recombinant DNA coding for said protein, (3) a vector having said DNA, (4) a transformant carrying said vector, and (5) a method for producing said protein wherein said transformant is cultured in a culture medium, the protein being useful not only in cloning other PGE receptor genes, clarifying the structure of PGE receptors and elucidating the function of PGE, but also in searching for PGE antagonists and agonists and so on.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 16 OF 21 USPATFULL on STN
ACCESSION NUMBER: 97:109877 USPATFULL
TITLE: Antisense oligonucleotides of human regulatory subunit RI-alpha of cAMP dependent protein kinases for the treatment of cancer
INVENTOR(S): Cho-Chung, Yoon S., 7017 Kenhill Rd., Bethesda, MD, United States 20817

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5691317		19971125
APPLICATION INFO.:	US 1995-383742		19950202 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-60984, filed on 14 May 1993 which is a division of Ser. No. US 1991-702163, filed on 20 May 1991, now patented, Pat. No. US 5271941, issued on 21 Dec 1993 which is a continuation-in-part of Ser. No. US 1991-680198, filed on 5 Apr 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-607113, filed on 2 Nov 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

PRIMARY EXAMINER: Rories, Charles C. P.
LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antisense oligonucleotides of human regulatory subunit RI-alpha of cAMP-dependent protein kinases are disclosed along with pharmaceutical compositions containing these oligonucleotides as the active ingredients. These antisense oligonucleotides are useful for inhibiting the growth of cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 17 OF 21 USPATFULL on STN
ACCESSION NUMBER: 97:38501 USPATFULL
TITLE: Antisense oligonucleotides of human regulatory subunit RI-.sub.α of camp dependent protein kinases for the treatment of cancer
INVENTOR(S): Cho-Chung, Yoon S., 7017 Kenhill Rd., Bethesda, MD, United States 20817

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5627158		19970506
APPLICATION INFO.:	US 1993-60984		19930514 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1991-702163, filed on 20 May 1991, now patented, Pat. No. US 5271941 which is a continuation-in-part of Ser. No. US 1991-680198, filed on 5 Apr 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-607113, filed on 2 Nov 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Rories, Charles C. P.
LEGAL REPRESENTATIVE: Sterne, Kessler Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1143
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antisense oligonucleotides of human regulatory subunit RI-alpha of cAMP-dependent protein kinases are disclosed along with pharmaceutical compositions containing these oligonucleotides as the active ingredients. These antisense oligonucleotides are useful for inhibiting the growth of cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 18 OF 21 USPATFULL on STN
ACCESSION NUMBER: 96:106365 USPATFULL
TITLE: Prostaglandin E receptors, their DNA and production
INVENTOR(S): Ichikawa, Atsushi, Osaka, Japan
Narumiya, Shuh, Kyoto, Japan
PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)

	NUMBER	KIND	DATE

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5576192		19961119
APPLICATION INFO.:	US 1995-390162		19950217 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-24179, filed on 23 Feb 1993, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1992-36580	19920224
	JP 1992-64889	19920323

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Draper, Garnette D.
ASSISTANT EXAMINER: Teng, Sally P.
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 987
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are (1) a protein capable of receiving PGE, (2) a recombinant DNA coding for said protein, (3) a vector having said DNA, (4) a transformant carrying said vector, and (5) a method for producing said protein wherein said transformant is cultured in a culture medium, the protein being useful not only in cloning other PGE receptor genes, clarifying the structure of PGE receptors and elucidating the function of PGE, but also in searching for PGE antagonists and agonists and so on.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 19 OF 21 USPATFULL on STN
ACCESSION NUMBER: 94:55470 USPATFULL
TITLE: DNA encoding rat and human protein kinase C
INVENTOR(S): Yoshitaka, Ono, Ikeda, Japan
Tsutomu, Kurokawa, Kawanishi, Japan
Koichi, Igarashi, Kyoto, Japan
Yasutomi, Nishizuka, Ashiya, Japan
PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5324651		19940628
APPLICATION INFO.:	US 1992-945739		19920916 (7)

RELATED APPLN. INFO.: Division of Ser. No. US 1991-654404, filed on 8 Feb 1991, now patented, Pat. No. US 5219748 which is a continuation of Ser. No. US 1987-65828, filed on 23 Jun 1987, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1986-149385	19860627
	JP 1986-217944	19860918
	JP 1986-281870	19861128
	JP 1987-40160	19870525

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Grimes, Eric
LEGAL REPRESENTATIVE: Conlin, David G., Resnick, David S.
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 17 Drawing Page(s)
LINE COUNT: 1020
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Human protein kinase C and rat protein kinase C, recombinant DNA containing DNA sequence coding for kinase C, a transformant transformed with a vector containing the above recombinant DNA, and the production method of human or rat kinase C by cultivating the transformant are disclosed.

Kinase C is useful as reagent for studying cellular signal transduction mechanism, as diagnostic and inspection agent for disease, for example tumor, resulting from the trouble of cellular signal transduction mechanism, and as screening agent for a preventive agent or medicine to the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 20 OF 21 USPATFULL on STN
ACCESSION NUMBER: 93:106813 USPATFULL
TITLE: Antisense oligonucleotides of human regulatory subunit RI.sub.α of cAMP-dependent protein kinases
INVENTOR(S): Cho-Chung, Yoon S., 7017 Kenhill Rd., Bethesda, MD, United States 20817

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5271941		19931221
APPLICATION INFO.:	US 1991-702163		19910520 (7)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1991-680198, filed on 5 Apr 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-607113, filed on 2 Nov 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Brown, Johnnie R.
ASSISTANT EXAMINER: Kunz, Gary L.
LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1007
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Antisense oligonucleotides of human regulatory subunit RI-alpha of cAMP-dependent protein kinases are disclosed along with pharmaceutical compositions containing these oligonucleotides as the active ingredients. These antisense oligonucleotides have been shown to inhibit the growth of several cancer cell lines including HL-60, human colon carcinoma LS-174T, neuroblastoma cells, breast cancer cells, and gastric carcinoma cells. In addition, these oligonucleotides can inhibit the growth of human colon carcinoma cells transplanted in athymic mice.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L29 ANSWER 21 OF 21 USPATFULL on STN
ACCESSION NUMBER: 93:48408 USPATFULL
TITLE: Recombinant human and rat protein kinase C polypetides
INVENTOR(S): Yoshitaka, Ono, Ikeda, Japan
Tsutomu, Kurokawa, Kawanishi, Japan
Koichi, Igarashi, Kyoto, Japan
Yasutomi, Nishizuka, Ashiya, Japan
PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5219748		19930615
APPLICATION INFO.:	US 1991-654404		19910208 (7)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1987-65828, filed on 23 Jun 1987

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1986-149385	19860627
	JP 1986-217944	19860918
	JP 1986-281870	19861128
	JP 1987-40160	19870525

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Moore, William W.
LEGAL REPRESENTATIVE: Conlin, David G., Resnick, David S.
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 17 Drawing Page(s)
LINE COUNT: 1022
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Human protein kinase C and rat protein kinase C, recombinant DNA containing DNA sequence coding for kinase C, a transformant transformed with a vector containing the above recombinant DNA, and the production method of human or rat kinase C by cultivating the transformant are disclosed.

Kinase C is useful as reagent for studying cellular signal transduction mechanism, as diagnostic and inspection agent for disease, for example tumor, resulting from the trouble of cellular signal transduction mechanism, and as screening agent for a preventive agent or medicine to the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

(FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO, USPATFULL' ENTERED AT 14:16:44 ON 14 MAR 2006) - Author(s)

L30	2011	SEA ABB=ON	PLU=ON	"MEYERS R"?/AU
L31	498	SEA ABB=ON	PLU=ON	("KAPELLER LIBERMANN R"? OR "LIBERMANN KAPELLER R"? OR "LIBERMANN R"? OR "KAPELLER R"?)/AU
L32	3809	SEA ABB=ON	PLU=ON	"WILLIAMSON M"?/AU
L33	45	SEA ABB=ON	PLU=ON	L30 AND L31 AND L32
L34	151	SEA ABB=ON	PLU=ON	L30 AND (L31 OR L32)
L35	65	SEA ABB=ON	PLU=ON	L31 AND L32
L36	47	SEA ABB=ON	PLU=ON	(L33 OR L34 OR L35) AND L14

Searcher : Shears 571-272-2528

L37 47 SEA ABB=ON PLU=ON L36 AND (PEPTIDE OR POLYPEPTIDE OR PROTEIN OR POLYPROTEIN)
L38 47 DUP REM L37 (0 DUPLICATES REMOVED)
L39 47 SEA ABB=ON PLU=ON L38 AND WASH?

L39 ANSWER 1 OF 47 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-351402 [38] WPIDS
DOC. NO. CPI: C2002-099715
TITLE: New isolated human nucleic acid molecules, designated 13237, 18480, 2245 or 16228, encode novel **protein kinase** family members, useful for the treatment and diagnosis of e.g. immune, cardiovascular, reproductive and cancerous disorders.
DERWENT CLASS: B04 D16
INVENTOR(S): **KAPELLER-LIBERMANN, R; MEYERS, R;** RUDOLPH-OWEN, L; TSAI, F; RUDOLPH-OWEN, L A; TSAI, F Y; BANDARU, R; HUNTER, J J; MACBETH, K J; **MEYERS, R E; WILLIAMSON, M J**
PATENT ASSIGNEE(S): (MILL-N) MILLENNIUM PHARM INC; (KAPE-I) KAPELLER-LIBERMANN R; (MEYE-I) MEYERS R; (RUDO-I) RUDOLPH-OWEN L A; (TSAI-I) TSAI F Y
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002006330	A2	20020124	(200238)*	EN	159
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001080639	A	20020130	(200241)		
US 2002068698	A1	20020606	(200241)		
EP 1335981	A2	20030820	(200362)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
US 2004033509	A1	20040219	(200414)		
AU 2001280639	A8	20051006	(200612)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2002006330	A2		WO 2001-US22820	20010718
AU 2001080639	A		AU 2001-80639	20010718
US 2002068698	A1	Provisional	US 2000-219028P	20000718
			US 2001-910150	20010718
EP 1335981	A2		EP 2001-959043	20010718
			WO 2001-US22820	20010718
US 2004033509	A1	Provisional	US 2000-219028P	20000718
		CIP of	US 2001-910150	20010718
		CIP of	US 2002-251507	20020920
			US 2003-377097	20030228
AU 2001280639	A8		AU 2001-280639	20010718

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001080639	A Based on	WO 2002006330
EP 1335981	A2 Based on	WO 2002006330
AU 2001280639	A8 Based on	WO 2002006330

PRIORITY APPLN. INFO: US 2000-219028P 20000718; US 2001-910150 20010718; US 2002-251507 20020920; US 2003-377097 20030228

AN 2002-351402 [38] WPIDS

AB WO 200206330 A UPAB: 20020618

NOVELTY - Isolated human nucleic acid molecules, designated 13237, 18480, 2245 or 16228, which encode novel **protein kinase** family members, are new.

DETAILED DESCRIPTION - Isolated human nucleic acid molecules, designated 13237, 18480, 2245 or 16228, which encode novel **protein kinase** family members, are new.

An isolated 13237, 18480, 2245 or 16228 nucleic acid molecule (N1) selected from:

(a) a nucleic acid comprising a sequence which is at least 60% or 100% identical to the 13237 (S1), 1066 (S2), 2438 (S3), 2079 (S4), 2245 (S5), 1278 (S6), 3301 (S7), or 2781 (S8) nucleotide sequence defined in the specification, or the nucleotide sequence (S9) of the DNA insert of the plasmid deposited with ATCC (Accession Number not given in the specification);

(b) a nucleic acid comprising a fragment of at least 15 nucleotides of a sequence selected from S1-S8 or S9;

(c) a nucleic acid which encodes a **polypeptide** comprising the 1066 (P1), 692 (P2), 425 (P3), 926 (P4) amino acid sequence, or the amino acid sequence encoded by the cDNA insert (C1) of the plasmid deposited with ATCC (Accession Number not defined in the specification);

(d) a nucleic acid molecule which encodes a fragment of a **polypeptide** comprising the amino acid sequence of P1-P4, or the amino acid sequence encoded by C1, where the fragment comprises at least 15 contiguous amino acids;

(e) a nucleic acid molecule which encodes a naturally occurring allelic variant of a **polypeptide** comprising the amino acid sequence of P1-P4, or the amino acid sequence encoded by C1, where the nucleic acid molecule **hybridizes** to a nucleic acid molecule comprising the sequence of S1-S8, or its complement under stringent conditions.

INDEPENDENT CLAIMS are included for the following:

(1) a host cell which contains N1;

(2) an isolated 13237, 18480, 2245 or 16228 **polypeptide** (P5) selected from:

(a) a **polypeptide** which is encoded by a nucleic acid molecule comprising a sequence which is at least 60% identical to a nucleic acid comprising the sequence of S1-S8, or S9, or its complement;

(b) a naturally occurring allelic variant of a **polypeptide** comprising the amino acid sequence of P1-P4, or the amino acid sequence encoded by C1, where the **polypeptide** is encoded by a nucleic acid molecule which **hybridizes** to a nucleic acid molecule comprising the sequence of S1-S8, or its complement under stringent conditions;

(c) a fragment of a **polypeptide** comprising the amino acid sequence of P1-P4, or the amino acid sequence encoded by C1, where the fragment comprises at least 15 contiguous amino acids; or

(d) the amino acid sequence of P1-P4;

(3) an antibody which selectively binds to P5;

(4) a method for producing the **polypeptide** of P5 comprising culturing the host cell of (1) under conditions in which the nucleic acid molecule is expressed;

(5) a method for detecting the presence of N1 or a **polypeptide** encoded by the nucleic acid molecule in a sample;

(6) a kit comprising a compound which selectively **hybridizes** to N1 or binds to a **polypeptide** encoded by the nucleic acid molecule and instructions for use;

(7) a method for identifying a compound which binds to a **polypeptide** or modulates the activity of P5, comprising;

(8) a method for modulating the activity of P5 comprising contacting the **polypeptide** or a cell expressing the **polypeptide** with a compound which binds to the **polypeptide** in a sufficient concentration to modulate the activity of the **polypeptide**;

(9) methods of identifying a nucleic acid molecule associated with cancer or a cellular proliferation and/or differentiation disorder;

(10) a method of identifying a **polypeptide** associated with cancer or a cellular proliferation and/or differentiation disorder;

(11) methods of identifying a subject having cancer or a cellular proliferation and/or differentiation disorder, or at risk for developing cancer or a cellular proliferation and/or differentiation disorder;

(12) a method for identifying a compound capable of treating cancer or a cellular proliferation and/or differentiation disorder, characterized by aberrant 13237, 18480, 2245 or 16228 nucleic acid expression or 13237, 18480, 2245 or 16228 **polypeptide** activity;

(13) a method (M1) for treating a subject having cancer or a cellular proliferation and/or differentiation disorder, or at risk of developing cancer or a cellular proliferation and/or differentiation disorder, comprising administering to the subject a modulator of N1 or the **polypeptide** encoded by the nucleic acid molecule, or contacting a cell with a 13237, 18480,2245 or 16228 modulator;

(14) a method for evaluating the efficacy of a treatment of cancer or a cellular proliferation and/or differentiation disorder in a subject; and

(15) a method of diagnosing cancer or a cellular proliferation and/or differentiation disorder, in a subject, comprising.

ACTIVITY - Immunomodulator; Antidiabetic; Antirheumatic; Antiarthritic; Antiinflammatory; Immunosuppressive; Endocrine-General; Hypertensive; Antiarteriosclerotic; Cardiant; Antiarrhythmic; Vasotropic; Antianginal; Antiallergic; Vulnerary; Neuroprotective; Tuberculostatic; Anti-HIV; Nootropic; Antiparkinsonian; Immunostimulant.

No biological data given.

MECHANISM OF ACTION - Antisense-Therapy; Angiogenesis-Stimulator; Angiogenesis-Inhibitor; Gene-Therapy

No biological data given.

USE - The proteins, polynucleotides, antibodies and modulators are useful in the treatment of cellular proliferative or differentiative, neural, cardiovascular, brain, prostatic, skin and

skeletal muscular disorders (claimed). They are also useful for the treatment and diagnosis of 13237, 18480, 2245 or 16228-mediated disorders e.g. protein-protein interaction disorders, signal transduction disorders, immune (e.g. diabetes, rheumatoid arthritis, SLE), reproductive, cardiovascular (e.g. hypertension, atherosclerosis, coronary artery disease, arrhythmias, ischemic heart disease, angina pectoris), vascular disorders (e.g. Wegeners granulomatosis, varicose veins, wound healing) or cancerous disorders, multiple sclerosis, Crohn's disease, ulcers, asthma, allergy, infection, hepatitis, kidney disease (e.g. glomerulonephritis), idiopathic thrombocytopenic purpura, tuberculosis, HIV, Alzheimer's; for the diagnosis of a predisposition to a disorder; for evaluating the efficacy of a therapeutic or prophylactic disorder; for chromosome mapping; as immunogens; for drug screening; for the detection of mutations in the gene; and for tissue typing.
Dwg.0/21

L39 ANSWER 2 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2006:15862 USPATFULL

TITLE: Novel nucleic acid sequences encoding adenylate kinase, phospholipid scramblase-like, DNA fragmentation factor-like, phosphatidylserine synthase-like, and ATPase-like molecules and uses therefor

INVENTOR(S): Chun, Miyoung, Belmont, MA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES

PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006014244	A1	20060119
APPLICATION INFO.:	US 2005-40595	A1	20050121 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2002-165800, filed on 7 Jun 2002, GRANTED, Pat. No. US 6953682
Continuation-in-part of Ser. No. US 2001-781677, filed on 12 Feb 2001, ABANDONED
Continuation-in-part of Ser. No. US 2001-795038, filed on 26 Feb 2001, ABANDONED
Continuation-in-part of Ser. No. US 2001-790180, filed on 21 Feb 2001, ABANDONED
Continuation-in-part of Ser. No. US 2001-790838, filed on 22 Feb 2001, GRANTED, Pat. No. US 6489152
Continuation-in-part of Ser. No. US 2001-790179, filed on 21 Feb 2001, GRANTED, Pat. No. US 6479268

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-181705P	20000210 (60)
	US 2000-186234P	20000229 (60)
	US 2000-185947P	20000229 (60)
	US 2000-185946P	20000229 (60)
	US 2000-185609P	20000229 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, CAMBRIDGE, MA, 02139, US
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 43 Drawing Page(s)
LINE COUNT: 18643
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules that encode novel **polypeptides**. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing the nucleic acid molecules of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a sequence of the invention has been introduced or disrupted. The invention still further provides isolated **proteins**, fusion **proteins**, antigenic **peptides** and antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 3 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2006:10778 USPATFULL
TITLE: Novel 27875, 22025, 27420, 17906, 16319, 55092 and 10218 molecules and uses therefor
INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES
White, David, Braintree, MA, UNITED STATES
Robison, Keith E., Wilmington, MA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Carroll, Joseph M., Cambridge, MA, UNITED STATES
Cook, William James, Hanover, NH, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES
Chun, Miyoung, Belmont, MA, UNITED STATES
Williamson, Mark J., Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006009632	A1	20060112
APPLICATION INFO.:	US 2005-226701	A1	20050914 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2003-386414, filed on 11 Mar 2003, PENDING Continuation-in-part of Ser. No. US 1999-426282, filed on 25 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-668266, filed on 22 Sep 2000, ABANDONED Division of Ser. No. US 1999-330970, filed on 11 Jun 1999, GRANTED, Pat. No. US 6146876 Continuation-in-part of Ser. No. US 2000-724599, filed on 28 Nov 2000, ABANDONED Continuation-in-part of Ser. No. US 2001-860193, filed on 16 May 2001, ABANDONED Division of Ser. No. US 2000-571689, filed on 16 May 2000, ABANDONED Continuation-in-part of Ser. No. US 2002-283023, filed on 29 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2001-10943, filed on 6 Dec 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-833082, filed on 10 Apr 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-335044P	20011031 (60)
	US 2000-254037P	20001207 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, CAMBRIDGE, MA, 02139, US
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 25188
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 27875, 22025, 27420, 16319, 55092 and 10218 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 27875, 22025, 27420, 16319, 55092 and 10218 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 27875, 22025, 27420, 16319, 55092 and 10218 gene has been introduced or disrupted. The invention still further provides isolated 27875, 22025, 27420, 17906, 16319, 55092 or 10218 **proteins**, fusion **proteins**, antigenic **peptides** and anti-27875, 22025, 27420, 17906, 16319, 55092 or 10218 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 4 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2006:3956 USPATFULL
TITLE: Novel human **protein kinase**, phosphatase, and protease family members and uses thereof
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Olandt, Peter J., Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Curtis, Rory A. J., Framingham, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
Weich, Nadine, Brookline, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006003413	A1	20060105
APPLICATION INFO.:	US 2005-151601	A1	20050613 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2002-170789, filed on 13 Jun 2002, PENDING Continuation-in-part of Ser. No. US 2001-797039, filed on 28 Feb 2001, GRANTED, Pat. No. US 6730491 Continuation-in-part of Ser. No. WO 2001-US6525, filed on 28 Feb 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US6525	20010228
	WO 2001-US19269	20010615
	WO 2001-US26052	20010821

WO 2001-US16549	20010521	
WO 2001-US7138	20010305	
WO 2001-US40483	20010411	
WO 2001-US29904	20010924	
WO 2001-US7074	20010305	
US 2000-186061P	20000229	(60)
US 2000-212078P	20000615	(60)
US 2000-226740P	20000821	(60)
US 2000-205508P	20000519	(60)
US 2000-197508P	20000418	(60)
US 2000-235023P	20000925	(60)
US 2000-246561P	20001107	(60)
US 2000-187454P	20000307	(60)
US 2000-187420P	20000307	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, CAMBRIDGE, MA, 02139, US
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 35 Drawing Page(s)
LINE COUNT: 9984
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 53070, 15985, 26583, 21953, m32404, 14089, and 23436 nucleic acid molecules, which encode novel human **protein kinase** family members, serine/threonine **protein kinase** family members, serine/threonine phosphatase family members, prolyl oligopeptidase family members, trypsin family members, trypsin serine protease family members, and ubiquitin protease family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 53070, 15985, 26583, 21953, m32404, 14089, or 23436 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 53070, 15985, 26583, 21953, m32404, 14089, or 23436 gene has been introduced or disrupted. The invention still further provides isolated 53070, 15985, 26583, 21953, m32404, 14089, or 23436 **proteins**, fusion **proteins**, antigenic **peptides** and anti-53070, 15985, 26583, 21953, m32404, 14089, or 23436 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 5 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2005:247698 USPATFULL
TITLE: Novel human genes and methods of use thereof
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Curtis, Rory A.J., Ashland, MA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 2005214893	A1	20050929
APPLICATION INFO.:	US 2004-968812	A1	20041019 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2002-176306, filed on 20 Jun 2002, ABANDONED Continuation-in-part of Ser. No. US 2001-1137, filed on 14 Nov 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US45291, filed on 14 Nov 2001, PENDING Continuation-in-part of Ser. No. US 2001-23617, filed on 18 Dec 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US49416, filed on 18 Dec 2001, PENDING Continuation-in-part of Ser. No. US 2001-83248, filed on 22 Oct 2001, ABANDONED

PRIORITY INFORMATION:	NUMBER	DATE
	WO 2001-US46717	20011022
	US 2000-248362P	20001114 (60)
	US 2000-248331P	20001114 (60)
	US 2000-248365P	20001114 (60)
	US 2000-250077P	20001130 (60)
	US 2000-250327P	20001130 (60)
	US 2000-250176P	20001130 (60)
	US 2000-256249P	20001218 (60)
	US 2000-256405P	20001218 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, CAMBRIDGE, MA, 02139, US
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 60 Drawing Page(s)
LINE COUNT: 26559
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 47476, 67210, 49875,46842,33201, 83378,84233,64708, 85041,84234,21617, 55562,23566,33489, and 57779 nucleic acid molecules, which encode novel human genes. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 gene has been introduced or disrupted. The invention still further provides isolated 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 **proteins**, fusion **proteins**, antigenic **peptides** and anti-47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 6 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2005:234110 USPATFULL
TITLE: 47169 and 33935, novel human glycosyl transferases and uses therefor

INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005203049	A1	20050915
APPLICATION INFO.:	US 2005-120749	A1	20050503 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-1851, filed on 20 Nov 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-249939P	20001120 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, Cambridge, MA, 02139, US
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1-35
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 5270
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 47169 and 33935 nucleic acid molecules, which encode novel glycosyl transferases. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 47169 and 33935 nucleic acid molecules, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a 47169 or 33935 gene has been introduced or disrupted. The invention still further provides isolated 47169 and 33935 **proteins**, fusion **proteins**, antigenic **peptides** and anti-47169 and anti-33935 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 7 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2005:23294 USPATFULL
TITLE: Novel human membrane-associated **protein** and cell surface **protein** family members
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Curtis, Rory A.J., Ashland, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
Leiby, Kevin R., Natick, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005019838	A1	20050127
APPLICATION INFO.:	US 2004-860779	A1	20040603 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2002-162435, filed on 4 Jun 2002, PENDING Continuation-in-part of Ser. No. US 2001-836499, filed on 17 Apr 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US12420, filed on 17 Apr 2001, PENDING Continuation-in-part of Ser. No. US 2001-891008, filed on 25 Jun 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US19963, filed on 25 Jun 2001, PENDING Continuation-in-part of Ser. No. US 2001-860868, filed on 18 May 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US16013, filed on 18 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-886429, filed on 21 Jun 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US20055, filed on 21 Jun 2001, PENDING Continuation-in-part of Ser. No. US 2002-41406, filed on 8 Jan 2002, ABANDONED Continuation-in-part of Ser. No. WO 2002-US275, filed on 8 Jan 2002, PENDING Continuation-in-part of Ser. No. US 2001-934268, filed on 21 Aug 2001, ABANDONED Continuation-in-part of Ser. No. WO 2001-US41811, filed on 21 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-197507P	20000418 (60)
	US 2000-214220P	20000623 (60)
	US 2000-205674P	20000519 (60)
	US 2000-213963P	20000623 (60)
	US 2001-260286P	20010108 (60)
	US 2000-226612P	20000821 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 40 Landsdowne Street, CAMBRIDGE, MA, 02139
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 30445
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 16051a, 16051b, 58199, 57805, 56739, 39362, and 23228 nucleic acid molecules, which encode novel human membrane-associated **protein** family members, and human cell surface **protein** family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 gene has been introduced or disrupted. The invention still further provides isolated 16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 **proteins**, fusion **proteins**, antigenic **peptides** and anti-16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Searcher : Shears 571-272-2528

L39 ANSWER 8 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:190234 USPATFULL
TITLE: 12832, a novel human kinase-like molecule and uses thereof
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Hodge, Martin R., Arlington, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004147004	A1	20040729
APPLICATION INFO.:	US 2003-682739	A1	20031009 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-562480, filed on 1 May 2000, GRANTED, Pat. No. US 6656698
Continuation-in-part of Ser. No. US 1999-345473, filed on 30 Jun 1999, GRANTED, Pat. No. US 6558903
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Intellectual Property Department, MILLENNIUM PHARMACEUTICALS, INC., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 3030
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel human kinase-like **polypeptides, proteins,** and nucleic acid molecules are disclosed. In addition to isolated, full-length kinase-like **proteins,** the invention further provides isolated kinase-like fusion **proteins,** antigenic **peptides,** and anti-kinase-like antibodies. The invention also provides kinase-like nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a kinase-like gene has been introduced or disrupted. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 9 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:158550 USPATFULL
TITLE: Novel 27877, 18080, 14081, 32140, 50352, 16658, 14223, 16002, 50566, 65552 and 65577 molecules and uses therefor
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Carroll, Joseph M., Cambridge, MA, UNITED STATES
Cook, William James, Hanover, NH, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Weich, Nadine S., Brookline, MA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 2004121349	A1	20040624
APPLICATION INFO.:	US 2003-391364	A1	20030318 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-950370, filed on 10 Sep 2001, ABANDONED Continuation-in-part of Ser. No. US 2002-294039, filed on 13 Nov 2002, PENDING Continuation-in-part of Ser. No. US 2002-266035, filed on 7 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2000-717926, filed on 21 Nov 2000, GRANTED, Pat. No. US 6569657 Continuation-in-part of Ser. No. US 2002-268036, filed on 9 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2001-922138, filed on 3 Aug 2001, PENDING Continuation-in-part of Ser. No. US 2001-945327, filed on 31 Aug 2001, PENDING Continuation-in-part of Ser. No. US 2002-163316, filed on 5 Jun 2002, PENDING Continuation-in-part of Ser. No. US 2002-103377, filed on 21 Mar 2002, PENDING

PRIORITY INFORMATION:	NUMBER	DATE
	US 2000-231084P	20000908 (60)
	US 2001-338587P	20011113 (60)
	US 2001-328198P	20011009 (60)
	US 2000-214707P	20000627 (60)
	US 2001-327820P	20011009 (60)
	US 2000-229299P	20000901 (60)
	US 2000-229425P	20000831 (60)
	US 2001-297863P	20010613 (60)
	US 2001-278347P	20010323 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Jean M. Silveri, Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 15849
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 27877, 18080, 14081, 32140, 50352, 16658, 14223, 16002, 50566, 65552 and 65577 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 27877, 18080, 14081, 32140, 50352, 16658, 14223, 16002, 50566, 65552 and 65577 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 27877, 18080, 14081, 32140, 50352, 16658, 14223, 16002, 50566, 65552 or 65577 gene has been introduced or disrupted. The invention still further provides isolated 27877, 18080, 14081, 32140, 50352, 16658, 14223, 16002, 50566, 65552 or 65577 **proteins**, fusion **proteins**, antigenic **peptides** and anti-27877, 18080, 14081, 32140, 50352, 16658, 14223, 16002, 50566, 65552 or 65577 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 10 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2004:109094 USPATFULL
TITLE: 18431 and 32374, novel human **protein kinase** family members and uses therefor
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Silos-Santiago, Immaculada, Cambridge, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004083496	A1	20040429
APPLICATION INFO.:	US 2003-678786	A1	20031003 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-916790, filed on 27 Jul 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-221543P	20000728 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Page(s)
LINE COUNT: 6026
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 32374 or 18431 nucleic acid molecules, which encode novel **protein kinase** family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 32374 or 18431 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 32374 or 18431 gene has been introduced or disrupted. The invention still further provides isolated 32374 or 18431 **proteins**, fusion **proteins**, antigenic **peptides** and anti-32374 or -18431 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 11 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:76577 USPATFULL
TITLE: Novel 21910, 56634, 55053, 2504, 15977, 14760, 25501, 17903, 3700, 21529, 26176, 26343, 56638, 18610, 33217, 21967, H1983, M1983, 38555 or 593 molecules and uses therefor
INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES
Hunter, John Joseph, Somerville, MA, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES
Rudolph-Owen, Laura A., Medford, MA, UNITED STATES
Curtis, Rory A. J., Framingham, MA, UNITED STATES
Olandt, Peter J., Newton, MA, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES

Galvin, Katherine M., Jamaica Plain, MA, UNITED STATES
Chun, Miyoung, Belmont, MA, UNITED STATES
Williamson, Mark J., Saugus, MA, UNITED STATES
Silos-Santiago, Inmaculada, Del Mar, CA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004058355	A1	20040325
APPLICATION INFO.:	US 2003-423543	A1	20030425 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-278036, filed on 22 Oct 2002, PENDING Continuation of Ser. No. US 2000-711216, filed on 9 Nov 2000, ABANDONED Continuation-in-part of Ser. No. US 2001-12055, filed on 13 Nov 2001, PENDING Continuation-in-part of Ser. No. US 2001-3690, filed on 15 Nov 2001, PENDING Continuation-in-part of Ser. No. US 2001-797039, filed on 28 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2002-217168, filed on 12 Aug 2002, PENDING Continuation-in-part of Ser. No. US 2001-929218, filed on 14 Aug 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-963159, filed on 25 Sep 2001, ABANDONED Continuation-in-part of Ser. No. US 2002-121911, filed on 12 Apr 2002, GRANTED, Pat. No. US 6607892 Division of Ser. No. US 1999-412210, filed on 5 Oct 1999, GRANTED, Pat. No. US 6403358 Continuation-in-part of Ser. No. US 2002-105989, filed on 25 Mar 2002, PENDING Continuation of Ser. No. US 1999-392189, filed on 9 Sep 1999, ABANDONED Continuation-in-part of Ser. No. US 2003-336153, filed on 3 Jan 2003, PENDING Continuation of Ser. No. US 2001-845044, filed on 27 Apr 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-928531, filed on 13 Aug 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-920346, filed on 31 Jul 2001, PENDING Continuation-in-part of Ser. No. US 2001-8016, filed on 8 Nov 2001, PENDING Continuation-in-part of Ser. No. US 2001-909743, filed on 20 Jul 2001, PENDING Division of Ser. No. US 1999-448076, filed on 23 Nov 1999, GRANTED, Pat. No. US 6300092 Continuation-in-part of Ser. No. US 1999-276400, filed on 25 Mar 1999, GRANTED, Pat. No. US 6140056 Continuation-in-part of Ser. No. US 2003-336489, filed on 2 Jan 2003, PENDING Continuation of Ser. No. US 2000-608921, filed on 30 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 1998-163821, filed on 30 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 2002-60763, filed on 30 Jan 2002, ABANDONED Continuation of Ser. No. US 1999-365162, filed on 30 Jul 1999, ABANDONED

NUMBER DATE

Searcher : Shears 571-272-2528

PRIORITY INFORMATION:	US 2000-205447P	20000519 (60)
	US 2000-248325P	20001114 (60)
	US 2000-248893P	20001115 (60)
	US 2000-186061P	20000229 (60)
	US 2001-312539P	20010815 (60)
	US 2000-257511P	20001222 (60)
	US 2000-234922P	20000925 (60)
	US 2000-200688P	20000428 (60)
	US 2000-235035P	20000925 (60)
	US 2000-221925P	20000731 (60)
	US 2001-260166P	20010105 (60)
	US 2000-246669P	20001108 (60)
	US 1999-117580P	19990127 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 14751
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 21910, 56634, 55053, 2504, 15977, 14760, 25501, 17903, 3700, 21529, 26176, 26343, 56638, 18610, 33217, 21967, h1983, m1983, 38555 and 593 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 21910, 56634, 55053, 2504, 15977, 14760, 25501, 17903, 3700, 21529, 26176, 26343, 56638, 18610, 33217, 21967, h1983, m1983, 38555 and 593 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 21910, 56634, 55053, 2504, 15977, 14760, 25501, 17903, 3700, 21529, 26176, 26343, 56638, 18610, 33217, 21967, h1983, m1983, 38555 or 593 gene has been introduced or disrupted. The invention still further provides isolated 21910, 56634, 55053, 2504, 15977, 14760, 25501, 17903, 3700, 21529, 26176, 26343, 56638, 18610, 33217, 21967, h1983, m1983, 38555 or 593 **proteins**, fusion **proteins**, antigenic **peptides** and anti-21910, 56634, 55053, 2504, 15977, 14760, 25501, 17903, 3700, 21529, 26176, 26343, 56638, 18610, 33217, 21967, h1983, m1983, 38555 or 593 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 12 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:50916 USPATFULL
TITLE: Novel human **protein kinases** and uses therefor
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004038346	A1	20040226

APPLICATION INFO.: US 2003-649156 A1 20030827 (10)
RELATED APPLN. INFO.: Division of Ser. No. US 2001-799875, filed on 6 Mar 2001, GRANTED, Pat. No. US 6638721
Continuation-in-part of Ser. No. US 2000-659289, filed on 12 Sep 2000, GRANTED, Pat. No. US 6518216

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-182059P	20000211 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 58 Drawing Page(s)
LINE COUNT: 6019
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to novel kinase nucleic acid sequences and **proteins**. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 13 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:44517 USPATFULL
TITLE: Novel 13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 and 32252 molecules and uses therefor
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Williamson, Mark J., Saugus, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Hunter, John Joseph, Somerville, MA, UNITED STATES
Rudolph-Owen, Laura A., Medford, MA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004033509	A1	20040219
APPLICATION INFO.:	US 2003-377097	A1	20030228 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-910150, filed on 18 Jul 2001, ABANDONED
Continuation-in-part of Ser. No. US 2002-251507, filed on 20 Sep 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-219028P	20000718 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 75 Sidney Street, Cambridge, MA, 02139

NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 15960
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 and 32252 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 and 32252 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 or 32252 gene has been introduced or disrupted. The invention still further provides isolated 13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 or 32252 **proteins**, fusion **proteins**, antigenic **peptides** and anti-13237, 18480, 2245, 16228, 7677, 26320, 46619, 33166, 16836, 46867, 21617, 55562, 39228, 62088, 46745, 23155, 21657, 42755, 32229, 22325, 46863 or 32252 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 14 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:13033 USPATFULL
TITLE: Novel 27411, 23413, 22438, 23553, 25278, 26212, NARC SC1, NARC 10A, NARC 1, NARC 12, NARC 13, NARC17, NARC 25, NARC 3, NARC 4, NARC 7, NARC 8, NARC 11, NARC 14A, NARC 15, NARC 16, NARC 19, NARC 20, NARC 26, NARC 27, NARC 28, NARC 30, NARC 5, NARC 6, NARC 9, NARC 10C, NARC 8B, NARC 9, NARC2A, NARC 16B, NARC 1C, NARC1A, NARC 25, 86604 and 32222 molecules and uses therefor
INVENTOR(S): Glucksmann, Maria A., Lexington, MA, UNITED STATES
Williamson, Mark J., Saugus, MA, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES
Rudolph-Owen, Laura A., Medford, MA, UNITED STATES
Kapeller-Libermann, Rosanna, Chestnut Hill, MA, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES
Chiang, Lillian Wei-Ming, Edison, NJ, UNITED STATES
Hunter, John Joseph, Somerville, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004009553	A1	20040115
APPLICATION INFO.:	US 2003-426776	A1	20030430 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-229662, filed on 28 Aug 2002, PENDING Division of Ser. No. US 2001-795691, filed on 28 Feb 2001, GRANTED, Pat. No. US 6465230 Continuation-in-part of Ser. No. US

2002-105992, filed on 25 Mar 2002, PENDING Continuation of Ser. No. US 1999-406045, filed on 27 Sep 1999, GRANTED, Pat. No. US 6451994 Continuation-in-part of Ser. No. US 2002-314881, filed on 9 Dec 2002, PENDING Continuation of Ser. No. US 2001-773426, filed on 31 Jan 2001, GRANTED, Pat. No. US 6534302 Continuation-in-part of Ser. No. US 2000-495823, filed on 31 Jan 2000, PENDING Continuation-in-part of Ser. No. US 2000-692785, filed on 20 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2002-284014, filed on 30 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2002-284059, filed on 30 Oct 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-185517P	20000228 (60)
	US 1999-161188P	19991022 (60)
	US 2001-335003P	20011031 (60)
	US 2001-335037P	20011031 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Jean M. Silveri, Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 24534
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules and **proteins**, designated 27411, 23413, 22438, 23553, 25278, 26212, NARC SC1, NARC 10A, NARC 1, NARC 12, NARC 13, NARC 17, NARC 25, NARC 3, NARC 4, NARC 7, NARC 8, NARC 11, NARC 14A, NARC 15, NARC 16, NARC 19, NARC 20, NARC 26, NARC 27, NARC 28, NARC 30, NARC 5, NARC 6, NARC 9, NARC 10C, NARC 8B, NARC 9, NARC2A, NARC 16B, NARC 1C, NARC 1A, NARC 25, 86604 and 32222 nucleic acid molecules and **proteins**. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing said nucleic acid molecules, host cells into which the expression vectors have been introduced, nonhuman transgenic animals in which a said genes have been introduced or disrupted, fusion **proteins**, antigenic **peptides** and antibodies to said **proteins** . Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 15 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2004:12981 USPATFULL
TITLE: Novel 25869, 25934, 26335, 50365, 21117, 38692, 46508, 16816, 16839, 49937, 49931 and 49933 molecules and uses therefor
INVENTOR(S): Curtis, Rory A. J., Ashland, MA, UNITED STATES
Logan, Thomas Joseph, Springfield, PA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES
Williamson, Mark J., Saugus, MA, UNITED

STATES
Rudolph-Owen, Laura A., Medford, MA, UNITED STATES
Chun, Miyoung, Belmont, MA, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004009501	A1	20040115
	US 2004157221	A9	20040812
APPLICATION INFO.:	US 2003-377072	A1	20030227 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-895860, filed on 29 Jun 2001, PENDING Continuation-in-part of Ser. No. US 2000-723806, filed on 28 Nov 2000, PENDING Continuation-in-part of Ser. No. US 2001-843297, filed on 25 Apr 2001, GRANTED, Pat. No. US 6569667 Continuation-in-part of Ser. No. US 2001-861801, filed on 21 May 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-816494, filed on 23 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2001-888911, filed on 25 Jun 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-908664, filed on 17 Jul 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-935291, filed on 21 Aug 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-215370P	20000629 (60)
	US 2000-187455P	20000307 (60)
	US 2000-199801P	20000426 (60)
	US 2000-205508P	20000519 (60)
	US 2000-213688P	20000623 (60)
	US 2000-218675P	20000717 (60)
	US 2000-250932P	20001130 (60)
	US 2000-226504P	20000821 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Jean M. Silveri, 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 16123
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 25869, 25934, 26335, 50365, 21117, 38692, 46508, 16816, 16839, 49937, 49931 and 49933 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 25869, 25934, 26335, 50365, 21117, 38692, 46508, 16816, 16839, 49937, 49931 or 49933 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 25869, 25934, 26335, 50365, 21117, 38692, 46508, 16816, 16839, 49937, 49931 or 49933 gene has been introduced or disrupted. The invention still further provides isolated 25869, 25934, 26335, 50365, 21117, 38692, 46508, 16816, 16839, 49937, 49931 or 49933 **proteins**, fusion **proteins**, antigenic **peptides** and anti-25869, 25934, 26335, 50365, 21117, 38692, 46508, 16816, 16839, 49937, 49931 or 49933 antibodies. Diagnostic and therapeutic methods utilizing

compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 16 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2004:7776 USPATFULL

TITLE: Novel 27875, 22025 ,27420, 17906, 16319, 55092 and 10218 molecules and uses therefor

INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES
White, David, Braintree, MA, UNITED STATES
Robison, Keith E., Wilmington, MA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Carroll, Joseph M., Cambridge, MA, UNITED STATES
Cook, William James, Hanover, NH, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES
Chun, Miyoung, Belmont, MA, UNITED STATES
Williamson, Mark J., Saugus, MA, UNITED STATES

PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004006016	A1	20040108
APPLICATION INFO.:	US 2003-386414	A1	20030311 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-426282, filed on 25 Oct 1999, ABANDONED
Continuation-in-part of Ser. No. US 2000-668266, filed on 22 Sep 2000, ABANDONED
Continuation-in-part of Ser. No. US 1999-330970, filed on 11 Jun 1999, GRANTED, Pat. No. US 6146876
Continuation-in-part of Ser. No. US 2000-724599, filed on 28 Nov 2000, PENDING Continuation-in-part of Ser. No. US 2001-860193, filed on 16 May 2001, PENDING Continuation-in-part of Ser. No. US 2000-571689, filed on 16 May 2000, ABANDONED
Continuation-in-part of Ser. No. US 2002-283023, filed on 29 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2001-10943, filed on 6 Dec 2001, PENDING Continuation-in-part of Ser. No. US 2001-833082, filed on 10 Apr 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-335044P	20011031 (60)
	US 2000-254037P	20001207 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 25349
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 27875, 22025, 27420, 16319, 55092 and 10218 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 27875, 22025, 27420,

16319, 55092 and 10218 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 27875, 22025, 27420, 16319, 55092 and 10218 gene has been introduced or disrupted. The invention still further provides isolated 27875, 22025, 27420, 17906, 16319, 55092 or 10218 **proteins**, fusion **proteins**, antigenic **peptides** and anti-27875, 22025, 27420, 17906, 16319, 55092 or 10218 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 17 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2004:7430 USPATFULL

TITLE: Novel 26199, 33530, 33949, 47148, 50226, 58764, 62113, 32144, 32235, 23565, 13305, 14911, 86216, 25206 and 8843 molecules and uses therefor

INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Curtis, Rory A. J., Ashland, MA, UNITED STATES
Rudolph-Owen, Laura A., Medford, MA, UNITED STATES
Weich, Nadine S., Brookline, MA, UNITED STATES
Olandt, Peter J., Buffalo, NY, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Carroll, Joseph M., Cambridge, MA, UNITED STATES

PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004005664	A1	20040108
APPLICATION INFO.:	US 2003-410764	A1	20030410 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-924358, filed on 6 Aug 2001, PENDING Continuation-in-part of Ser. No. US 2003-350553, filed on 24 Jan 2003, PENDING Continuation-in-part of Ser. No. US 2001-966614, filed on 27 Sep 2001, PENDING Continuation-in-part of Ser. No. US 2002-281094, filed on 25 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2002-76535, filed on 15 Feb 2002, PENDING Continuation-in-part of Ser. No. US 2001-860352, filed on 17 May 2001, ABANDONED Continuation-in-part of Ser. No. US 2000-593927, filed on 15 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2002-226410, filed on 23 Aug 2002, PENDING Continuation-in-part of Ser. No. US 2001-997816, filed on 29 Nov 2001, ABANDONED Continuation-in-part of Ser. No. US 2000-686673, filed on 11 Oct 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-229300P	20000901 (60)
	US 2002-351572P	20020124 (60)
	US 2000-238054P	20001005 (60)
	US 2001-347815P	20011029 (60)
	US 2001-269440P	20010216 (60)

	US 2000-205301P 20000519 (60)
	US 2000-199391P 20000425 (60)
	US 2001-314884P 20010824 (60)
	US 2000-250186P 20001130 (60)
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Jean M. Silveri, Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS:	19
EXEMPLARY CLAIM:	1
LINE COUNT:	17049

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 26199, 33530, 33949, 47148, 50226, 58764, 62113, 32144, 32235, 23565, 13305, 14911, 86216, 25206 and 8843 nucleic acid molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 26199, 33530, 33949, 47148, 50226, 58764, 62113, 32144, 32235, 23565, 13305, 14911, 86216, 25206 and 8843 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 26199, 33530, 33949, 47148, 50226, 58764, 62113, 32144, 32235, 23565, 13305, 14911, 86216, 25206 or 8843 gene has been introduced or disrupted. The invention still further provides isolated 26199, 33530, 33949, 47148, 50226, 58764, 62113, 32144, 32235, 23565, 13305, 14911, 86216, 25206 or 8843 **proteins**, fusion **proteins**, antigenic **peptides** and anti-26199, 33530, 33949, 47148, 50226, 58764, 62113, 32144, 32235, 23565, 13305, 14911, 86216, 25206 or 8843 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 18 OF 47 USPATFULL on STN

ACCESSION NUMBER:	2003:318673 USPATFULL
TITLE:	14400, 2838, 14618, 15334, 14274, 32164, 39404, 38911, 26904, 31237, 18057, 16405, 32705, 23224, 27423, 32700, 32712 and 12216, novel seven-transmembrane **proteins**/G-**protein** coupled receptors
INVENTOR(S):	Glucksmann, Maria A., Lexington, MA, UNITED STATES Weich, Nadine S., Brookline, MA, UNITED STATES Hunter, John Joseph, Somerville, MA, UNITED STATES White, David, Braintree, MA, UNITED STATES MacBeth, Kyle J., Boston, MA, UNITED STATES **Williamson, Mark J.**, Saugus, MA, UNITED STATES **Meyers, Rachel E.**, Newton, MA, UNITED STATES Chun, Miyoung, Belmont, MA, UNITED STATES
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003224417	A1	20031204
APPLICATION INFO.:	US 2003-400991	A1	20030327 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2002-190469, filed on 5 Jul 2002, PENDING Continuation of Ser. No. US 1999-439159, filed on 12 Nov 1999, ABANDONED		

Division of Ser. No. US 1998-137063, filed on 20 Aug 1998, ABANDONED Continuation-in-part of Ser. No. US 2002-167192, filed on 11 Jun 2002, PENDING Division of Ser. No. US 1999-420187, filed on 18 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 1998-173869, filed on 16 Oct 1998, ABANDONED Continuation-in-part of Ser. No. US 2003-339056, filed on 9 Jan 2003, PENDING Continuation of Ser. No. US 1999-377429, filed on 19 Aug 1999, ABANDONED Continuation-in-part of Ser. No. US 1998-136726, filed on 19 Aug 1998, ABANDONED Continuation-in-part of Ser. No. US 2001-911583, filed on 24 Jul 2001, ABANDONED Continuation-in-part of Ser. No. US 1999-476287, filed on 30 Dec 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-475790, filed on 30 Dec 1999, ABANDONED Continuation-in-part of Ser. No. US 2001-779448, filed on 8 Feb 2001, ABANDONED Continuation-in-part of Ser. No. US 1999-347094, filed on 2 Jul 1999, ABANDONED Continuation-in-part of Ser. No. US 2001-794257, filed on 27 Feb 2001, PENDING Continuation-in-part of Ser. No. US 1999-448687, filed on 24 Nov 1999, PENDING Continuation-in-part of Ser. No. US 1998-200302, filed on 25 Nov 1998, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-180986P	20000208 (60)
	US 2000-185606P	20000229 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Jean M. Silveri, Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 10269
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to a newly identified receptor belonging to the superfamily of G-**protein**-coupled receptors. The invention also relates to polynucleotides encoding the receptor. The invention further relates to methods using the receptor **polypeptides** and polynucleotides as a target for diagnosis and treatment in receptor-mediated disorders. The invention further relates to drug-screening methods using the receptor **polypeptides** and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the receptor **polypeptides** and polynucleotides. The invention further relates to procedures for producing the receptor **polypeptides** and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 19 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2003:318632 USPATFULL
TITLE: Novel human transferase family members and uses thereof

INVENTOR(S): Meyers, Rachel E., Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
Leiby, Kevin R., Natick, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Olandt, Peter J., Newton, MA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Rudolph-Owen, Laura A., Jamaica Plain, MA, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES
Hunter, John J., Somerville, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003224376	A1	20031204
APPLICATION INFO.:	US 2002-184648	A1	20020627 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-815028, filed on 22 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2001-801220, filed on 7 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2001-816714, filed on 23 Mar 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-844948, filed on 27 Apr 2001, PENDING Continuation-in-part of Ser. No. US 2001-861164, filed on 18 May 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-883060, filed on 15 Jun 2001, PENDING Continuation-in-part of Ser. No. US 2001-962678, filed on 25 Sep 2001, PENDING Continuation-in-part of Ser. No. US 2001-973457, filed on 9 Oct 2001, PENDING Continuation-in-part of Ser. No. US 2002-72285, filed on 8 Feb 2002, PENDING Continuation-in-part of Ser. No. US 2001-817910, filed on 26 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2001-842528, filed on 25 Apr 2001, ABANDONED Continuation-in-part of Ser. No. US 2001-882836, filed on 15 Jun 2001, PENDING Continuation-in-part of Ser. No. US 2001-882872, filed on 15 Jun 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US9358	20010322
	WO 2001-US7269	20010307
	WO 2001-US9468	20010323
	WO 2001-US13805	20010427
	WO 2001-US16292	20010518
	WO 2001-US19138	20010615
	WO 2001-US29963	20010925
	WO 2002-US3736	20020208
	WO 2001-US9633	20010326
	WO 2001-US40607	20010425
	WO 2001-US19543	20010615
	WO 2001-US19153	20010615
	US 2000-191964P	20000324 (60)
	US 2000-187456P	20000307 (60)
	US 2000-191865P	20000324 (60)
	US 2000-200604P	20000428 (60)
	US 2000-205408P	20000519 (60)
	US 2000-212079P	20000615 (60)

	US 2000-235044P	20000925 (60)
	US 2000-238849P	20001006 (60)
	US 2001-267494P	20010208 (60)
	US 2000-192092P	20000324 (60)
	US 2000-199500P	20000425 (60)
	US 2000-211730P	20000615 (60)
	US 2000-212077P	20000615 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Theodore R. Allen, Millennium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139	
NUMBER OF CLAIMS:	19	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	125 Drawing Page(s)	
LINE COUNT:	66695	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 33877, 47179, 26886, 25552, 32132, 32244, 23680, 32624, 47174, 60491, 46743, 27417, 27960, 32252, and 53320 nucleic acid molecules, which encode novel human transferase family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 33877, 47179, 26886, 25552, 32132, 32244, 23680, 32624, 47174, 60491, 46743, 27417, 27960, 32252, or 53320 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 33877, 47179, 26886, 25552, 32132, 32244, 23680, 32624, 47174, 60491, 46743, 27417, 27960, 32252, or 53320 gene has been introduced or disrupted. The invention still further provides isolated 33877, 47179, 26886, 25552, 32132, 32244, 23680, 32624, 47174, 60491, 46743, 27417, 27960, 32252, or 53320 proteins, fusion proteins, antigenic peptides and anti-33877, 47179, 26886, 25552, 32132, 32244, 23680, 32624, 47174, 60491, 46743, 27417, 27960, 32252, or 53320 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 20 OF 47 USPATFULL on STN

ACCESSION NUMBER:	2003:314555 USPATFULL
TITLE:	12832, a novel human kinase-like molecule and uses thereof
INVENTOR(S):	**Meyers, Rachel**, Newton, MA, United States Hodge, Martin R., Arlington, MA, United States **Williamson, Mark**, Saugus, MA, United States
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6656698	B1	20031202
APPLICATION INFO.:	US 2000-562480		20000501 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1999-345473, filed on 30 Jun 1999, now patented, Pat. No. US 6558903		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	GRANTED		
PRIMARY EXAMINER:	Monshipouri, Maryam		
LEGAL REPRESENTATIVE:	Millennium Pharmaceuticals, Inc.		

NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 3031
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novel human kinase-like **polypeptides, proteins,** and nucleic acid molecules are disclosed. In addition to isolated, full-length kinase-like **proteins,** the invention further provides isolated kinase-like fusion **proteins,** antigenic **peptides,** and anti-kinase-like antibodies. The invention also provides kinase-like nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a kinase-like gene has been introduced or disrupted. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 21 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2003:306464 USPATFULL
TITLE: Methods for using 22045, a human cyclic nucleotide phosphodiesterase
INVENTOR(S): **Kapeller-Libermann, Rosana,** Chestnut Hill, MA, UNITED STATES
Hunter, John Joseph, Somerville, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003215898	A1	20031120
APPLICATION INFO.:	US 2003-458839	A1	20030611 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-420190, filed on 18 Oct 1999, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS, INC., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 3727
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to methods for using a human cyclic nucleotide phosphodiesterase belonging to the superfamily of mammalian phosphodiesterases. The invention also relates to methods for using polynucleotides encoding the phosphodiesterase. The invention relates to methods using the phosphodiesterase **polypeptides** and polynucleotides as a target for diagnosis and treatment in phosphodiesterase-mediated or -related disorders. The invention further relates to drug-screening methods using the phosphodiesterase **polypeptides** and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the phosphodiesterase **polypeptides** and polynucleotides. The invention further relates to agonists and antagonists identifiefd by

drug screening methods with the phosphodiesterase **polypeptides** and polynucleotides as a target.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 22 OF 47 USPATFULL on STN

ACCESSION NUMBER:	2003:257879 USPATFULL
TITLE:	Novel human protein kinase, phosphatase, and protease family members and uses thereof
INVENTOR(S):	Meyers, Rachel E., Newton, MA, UNITED STATES Olandt, Peter J., Newton, MA, UNITED STATES Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES Curtis, Rory A. J., Framingham, MA, UNITED STATES Williamson, Mark, Saugus, MA, UNITED STATES Weich, Nadine, Brookline, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003180930	A1	20030925
APPLICATION INFO.:	US 2002-170789	A1	20020613 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-797039, filed on 28 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-882166, filed on 15 Jun 2001, PENDING Continuation-in-part of Ser. No. US 2001-934406, filed on 21 Aug 2001, PENDING Continuation-in-part of Ser. No. US 2001-861801, filed on 21 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-801267, filed on 6 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2001-829671, filed on 10 Apr 2001, PENDING Continuation-in-part of Ser. No. US 2001-961721, filed on 24 Sep 2001, PENDING Continuation-in-part of Ser. No. US 2001-45367, filed on 7 Nov 2001, PENDING Continuation-in-part of Ser. No. US 2001-801275, filed on 6 Mar 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US6525	20010228
	WO 2001-US19269	20010615
	WO 2001-US26052	20010821
	WO 2001-US16549	20010521
	WO 2001-US7138	20010305
	WO 2001-US40483	20010411
	WO 2001-US29904	20010924
	WO 2001-US7074	20010305
	US 2000-186061P	20000229 (60)
	US 2000-212078P	20000615 (60)
	US 2000-226740P	20000821 (60)
	US 2000-205508P	20000519 (60)
	US 2000-187454P	20000307 (60)
	US 2000-197508P	20000418 (60)
	US 2000-235023P	20000925 (60)
	US 2000-246561P	20001107 (60)
	US 2000-187420P	20000307 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	LOUIS MYERS, Fish & Richardson P.C., 225 Franklin

Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 62 Drawing Page(s)
LINE COUNT: 45159
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 2504, 15977, 14760, 53070, 15985, 50365, 26583, 21953, m32404, 14089, and 23436 nucleic acid molecules, which encode novel human protein kinase family members, serine/threonine protein kinase family members, hexokinase family members, serine/threonine phosphatase family members, prolyl oligopeptidase family members, trypsin family members, trypsin serine protease family members, and ubiquitin protease family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 2504, 15977, 14760, 53070, 15985, 50365, 26583, 21953, m32404, 14089, or 23436 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 2504, 15977, 14760, 53070, 15985, 50365, 26583, 21953, m32404, 14089, or 23436 gene has been introduced or disrupted. The invention still further provides isolated 2504, 15977, 14760, 53070, 15985, 50365, 26583, 21953, m32404, 14089, or 23436 proteins, fusion proteins, antigenic peptides and anti-2504, 15977, 14760, 53070, 15985, 50365, 26583, 21953, m32404, 14089, or 23436 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 23 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2003:188692 USPATFULL
TITLE: Novel human genes and methods of use thereof
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Curtis, Rory A. J., Framingham, MA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003130485	A1	20030710
APPLICATION INFO.:	US 2002-176306	A1	20020620 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-1137, filed on 14 Nov 2001, PENDING Continuation-in-part of Ser. No. WO 2001-US45291, filed on 14 Nov 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US49416	20011218
	WO 2001-US46717	20011022
	US 2000-248362P	20001114 (60)
	US 2000-248331P	20001114 (60)
	US 2000-248365P	20001114 (60)
	US 2000-250077P	20001130 (60)
	US 2000-250327P	20001130 (60)

	US 2000-250176P 20001130 (60)
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	LOUIS MYERS, Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS:	19
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	60 Drawing Page(s)
LINE COUNT:	26835

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, and 57779 nucleic acid molecules, which encode novel human genes. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 gene has been introduced or disrupted. The invention still further provides isolated 47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 **proteins**, fusion **proteins**, antigenic **peptides** and anti-47476, 67210, 49875, 46842, 33201, 83378, 84233, 64708, 85041, 84234, 21617, 55562, 23566, 33489, or 57779 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 24 OF 47 USPATFULL on STN

ACCESSION NUMBER:	2003:165934 USPATFULL
TITLE:	26343, a novel human oxidoreductase and uses therefor
INVENTOR(S):	**Meyers, Rachel**, Newton, MA, UNITED STATES **Williamson, Mark**, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S):	MILLENNIUM PHARMACEUTICALS, INC., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003113790	A1	20030619
APPLICATION INFO.:	US 2003-336153	A1	20030103 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-845044, filed on 27 Apr 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-200688P	20000428 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS:	31
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	7 Drawing Page(s)
LINE COUNT:	4775

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated OP nucleic acid molecules, which encode a novel family of oxidoreductase **proteins**. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing OP nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an OP gene has been introduced or disrupted. The invention still further provides isolated OP **proteins**, fusion **proteins**, antigenic **peptides**, and anti-OP antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 25 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2003:140464 USPATFULL

TITLE: Novel human membrane-associated **protein** and cell surface **protein** family members

INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Curtis, Rory A. J., Framingham, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Bandaru, Rajasekhar, Watertown, MA, UNITED STATES
Leiby, Kevin R., Natick, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003096305	A1	20030522
APPLICATION INFO.:	US 2002-162435	A1	20020604 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-836499, filed on 17 Apr 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US12420	20010417
	WO 2001-US19963	20010625
	WO 2001-US16013	20010518
	WO 2001-US20055	20010621
	WO 2002-US275	20020108
	WO 2001-US41811	20010821
	US 2000-197507P	20000418 (60)
	US 2000-214220P	20000623 (60)
	US 2000-205674P	20000519 (60)
	US 2000-213963P	20000623 (60)
	US 2001-260286P	20010108 (60)
	US 2000-226612P	20000821 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 30445

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 16051a, 16051b, 58199, 57805, 56739, 39362, and 23228 nucleic acid molecules, which encode novel human membrane-associated **protein** family members, and human cell surface **protein** family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 gene has been introduced or disrupted. The invention still further provides isolated 16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 **proteins**, fusion **proteins**, antigenic **peptides** and anti-16051a, 16051b, 58199, 57805, 56739, 39362, or 23228 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 26 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2003:134031 USPATFULL

TITLE: Novel nucleic acid sequences encoding adenylate kinase, phospholipid scramblase-like, DNA fragmentation factor-like, phosphatidylserine synthase-like, and ATPase-like molecules and uses therefor

INVENTOR(S): Chun, Miyoung, Belmont, MA, UNITED STATES
Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Meyers, Rachel E., Newton, MA, UNITED STATES

PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003092116	A1	20030515
	US 6953682	B2	20051011
APPLICATION INFO.:	US 2002-165800	A1	20020607 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-781677, filed on 12 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-795038, filed on 26 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-790180, filed on 21 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-790838, filed on 22 Feb 2001, GRANTED, Pat. No. US 6489152 Continuation-in-part of Ser. No. US 2001-790179, filed on 21 Feb 2001, GRANTED, Pat. No. US 6479268

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-181705P	20000210 (60)
	US 2000-186234P	20000229 (60)
	US 2000-185947P	20000229 (60)
	US 2000-185946P	20000229 (60)
	US 2000-185609P	20000229 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 43 Drawing Page(s)
LINE COUNT: 18760
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules that encode novel **polypeptides**. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing the nucleic acid molecules of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a sequence of the invention has been introduced or disrupted. The invention still further provides isolated **proteins**, fusion **proteins**, antigenic **peptides** and antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 27 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2003:86313 USPATFULL
TITLE: Novel human 39228, 21956, 25856, 22244, 8701, 32263, 50250, 55158, 47765, 62088, 50566, and 48118 molecules and uses therefor
INVENTOR(S): Meyers, Rachel E., Newton, MA, UNITED STATES
Rudolph-Owen, Laura A., Jamaica Plain, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA, UNITED STATES, 02139 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059919	A1	20030327
APPLICATION INFO.:	US 2002-160501	A1	20020530 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-838573, filed on 18 Apr 2001, PENDING Continuation-in-part of Ser. No. US 2001-870133, filed on 29 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-870130, filed on 29 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-862535, filed on 21 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-870383, filed on 29 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-860821, filed on 18 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-870110, filed on 29 May 2001, PENDING Continuation-in-part of Ser. No. US 2001-907509, filed on 16 Jul 2001, PENDING Continuation-in-part of Ser. No. US 2001-945327, filed on 31 Aug 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-197747P	20000418 (60)
	US 2000-207649P	20000526 (60)
	US 2000-207640P	20000526 (60)
	US 2000-205961P	20000519 (60)

	US 2000-207506P	20000526 (60)
	US 2000-205449P	20000519 (60)
	US 2000-207650P	20000526 (60)
	US 2000-218385P	20000714 (60)
	US 2000-229425P	20000831 (60)
	US 2001-318581P	20010910 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109	
NUMBER OF CLAIMS:	23	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	100 Drawing Page(s)	
LINE COUNT:	44311	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 39228, 21956, 25856, 22244, 8701, 32263, 50250, 55158, 47765, 62088, 50566, and 48118 nucleic acid molecules, which encode novel GTPase activating molecules, cadherin molecules, and ankyrin containing family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 39228, 21956, 25856, 22244, 8701, 32263, 50250, 55158, 47765, 62088, 50566, and 48118 nucleic acid molecules, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a 39228, 21956, 25856, 22244, 8701, 32263, 50250, 55158, 47765, 62088, 50566, or 48118 gene has been introduced or disrupted. The invention still further provides isolated 39228, 21956, 25856, 22244, 8701, 32263, 50250, 55158, 47765, 62088, 50566, and 48118 polypeptides, fusion polypeptides, antigenic peptides and anti-39228, 21956, 25856, 22244, 8701, 32263, 50250, 55158, 47765, 62088, 50566, and 48118 antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 28 OF 47 USPATFULL on STN

ACCESSION NUMBER:	2003:52389 USPATFULL
TITLE:	Novel nucleic acid sequences encoding a human ubiquitin protease, lipase, dynamin, short chain dehydrogenase, and ADAM-TS metalloprotease and uses therefor
INVENTOR(S):	Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES **Meyers, Rachel E.**, Newton, MA, UNITED STATES Rudolph-Owen, Laura A., Jamaica Plain, MA, UNITED STATES
PATENT ASSIGNEE(S):	Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003037350	A1	20030220
APPLICATION INFO.:	US 2002-163547	A1	20020605 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1999-407356, filed on 29 Sep 1999, PENDING Continuation-in-part of Ser. No. US 2000-704918, filed on 2 Nov 2000, PENDING Continuation-in-part of Ser. No. US		

1999-435311, filed on 5 Nov 1999, PENDING Continuation-in-part of Ser. No. US 2001-796100, filed on 28 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-781598, filed on 12 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-782952, filed on 14 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2000-496005, filed on 1 Feb 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-185503P	20000228 (60)
	US 2000-182009P	20000211 (60)
	US 2000-182408P	20000214 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MILLENNIUM PHARMACEUTICALS GROUP, Intellectual Property Group, 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 55 Drawing Page(s)
LINE COUNT: 23031
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules that encode novel **polypeptides**. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing the nucleic acid molecules of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a sequence of the invention has been introduced or disrupted. The invention still further provides isolated **proteins**, fusion **proteins**, antigenic **peptides** and antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 29 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2003:10675 USPATFULL
TITLE: 13295 novel **protein kinase** molecules and uses therefor
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003008370	A1	20030109
APPLICATION INFO.:	US 2002-172088	A1	20020613 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-596071, filed on 16 Jun 2000, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-199391P	20000425 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MORRISON & FOERSTER LLP, 3811 VALLEY CENTRE DRIVE, SUITE 500, SAN DIEGO, CA, 92130-2332

NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Page(s)
LINE COUNT: 3344
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated 13295 nucleic acid molecules, which encode novel **protein kinases**. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 13295 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 13295 gene has been introduced or disrupted. The invention still further provides isolated 13295 **proteins**, fusion **proteins**, antigenic **peptides** and anti-13295 antibodies. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 30 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2003:3442 USPATFULL
TITLE: 26176, a novel calpain protease and uses thereof
INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003003477	A1	20030102
APPLICATION INFO.:	US 2002-105989	A1	20020325 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-392189, filed on 9 Sep 1999, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 3443
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel calpain protease **polypeptides**, **proteins**, and nucleic acid molecules are disclosed. In addition to isolated, full-length calpain protease **proteins**, the invention further provides isolated calpain protease fusion **proteins**, antigenic **peptides**, and anti-calpain protease antibodies. The invention also provides calpain protease nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a calpain protease gene has been introduced or disrupted. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 31 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2002:346807 USPATFULL
TITLE: 33167, a novel human hydrolase and uses therefor
INVENTOR(S): Glucksmann, Maria Alexandra, Lexington, MA, United States
Meyers, Rachel, Newton, MA, United States
Williamson, Mark, Saugus, MA, United States
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6500657	B1	20021231
APPLICATION INFO.:	US 2000-584568		20000531 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193954P	20000331 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Patterson, Jr., Charles L.
LEGAL REPRESENTATIVE: Lahive & Cockfield, LLP, Mandragouras, Amy E., Laccotripe, Maria C.
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Figure(s); 12 Drawing Page(s)
LINE COUNT: 4336
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated HYDL-1 nucleic acid molecules, which encode novel hydrolase molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing HYDL-1 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an HYDL-1 gene has been introduced or disrupted. The invention still further provides isolated HYDL-1 **proteins**, fusion **proteins**, antigenic **peptides** and anti-HYDL-1 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 32 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:307896 USPATFULL
TITLE: 26886, a novel carnitine acyltransferase family member and uses therefor
INVENTOR(S): **Meyers, Rachel A.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002173020	A1	20021121
APPLICATION INFO.:	US 2001-801220	A1	20010307 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-187456P	20000307 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, FISH & RICHARDSON P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 5347
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated 26886 nucleic acid molecules, which encode a novel carnitine acyltransferase. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 26886 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 26886 gene has been introduced or disrupted. The invention still further provides isolated 26886 **proteins**, fusion **proteins**, antigenic **peptides** and anti-26886 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 33 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:280814 USPATFULL
TITLE: 22012, a novel human carboxypeptidase
INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES
MacBeth, Kyle J., Boston, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002156264	A1	20021024
APPLICATION INFO.:	US 2002-68134	A1	20020206 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-345469, filed on 30 Jun 1999, GRANTED, Pat. No. US 6369210
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 3398
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a newly identified human carboxypeptidase. The invention also relates to polynucleotides encoding the carboxypeptidase. The invention further relates to methods using the carboxypeptidase **polypeptides** and polynucleotides as a target for diagnosis and treatment in carboxypeptidase-related disorders. The invention further relates to drug-screening methods using the carboxypeptidase **polypeptides** and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the carboxypeptidase **polypeptides** and polynucleotides. The invention further relates to procedures for producing the carboxypeptidase

polypeptides and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 34 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:251226 USPATFULL
TITLE: 33167, a novel human hydrolase and uses therefor
INVENTOR(S): Glucksmann, Maria Alexandra, Lexington, MA, UNITED STATES
Meyers, Rachel, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002137172	A1	20020926
APPLICATION INFO.:	US 2002-80644	A1	20020222 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-584568, filed on 31 May 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-193954P	20000331 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 58
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 4047
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated HYDL-1 nucleic acid molecules, which encode novel hydrolase molecules. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing HYDL-1 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an HYDL-1 gene has been introduced or disrupted. The invention still further provides isolated HYDL-1 **proteins**, fusion **proteins**, antigenic **peptides** and anti-HYDL-1 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 35 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:221327 USPATFULL
TITLE: 46863, a novel human methyltransferase and uses thereof
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
Rudolph-Owen, Laura A., Jamaica Plain, MA, UNITED STATES

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 2002119466	A1	20020829
APPLICATION INFO.:	US 2001-939521	A1	20010824 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-227867P	20000824 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 5291
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acid molecules, designated TPRM nucleic acid molecules, which encode novel methyltransferase family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing TPRM nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a TPRM gene has been introduced or disrupted. The invention still further provides isolated TPRM **proteins**, fusion **proteins**, antigenic **peptides** and anti-TPRM antibodies. Diagnostic and therapeutic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 36 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:214240 USPATFULL
TITLE: 47169 and 33935, novel human glycosyl transferases and uses thereof
INVENTOR(S): **Meyers, Rachel E.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA, UNITED STATES, 02139 (2)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002115628	A1	20020822
APPLICATION INFO.:	US 2001-1851	A1	20011120 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-249939P	20001120 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 5365
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated

47169 and 33935 nucleic acid molecules, which encode novel glycosyl transferases. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 47169 and 33935 nucleic acid molecules, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a 47169 or 33935 gene has been introduced or disrupted. The invention still further provides isolated 47169 and 33935 **proteins**, fusion **proteins**, antigenic **peptides** and anti-47169 and anti-33935 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 37 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:157034 USPATFULL
TITLE: METHODS FOR USING 22045, A HUMAN CYCLIC NUCLEOTIDE PHOSPHODIESTERASE
INVENTOR(S): **KAPELLER-LIBERMANN, ROSANA**, CHESTNUT HILL, MA, UNITED STATES
HUNTER, JOHN JOSEPH, SOMERVILLE, MA, UNITED STATES
WILLIAMSON, MARK, SAUGUS, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002081633	A1	20020627	
	US 6673564	B2	20040106	
APPLICATION INFO.:	US 1999-420190	A1	19991018	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 4212
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods for using a human cyclic nucleotide phosphodiesterase belonging to the superfamily of mammalian phosphodiesterases. The invention also relates to methods for using polynucleotides encoding the phosphodiesterase. The invention relates to methods using the phosphodiesterase **polypeptides** and polynucleotides as a target for diagnosis and treatment in phosphodiesterase-mediated or -related disorders. The invention further relates to drug-screening methods using the phosphodiesterase **polypeptides** and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the phosphodiesterase **polypeptides** and polynucleotides. The invention further relates to agonists and antagonists identifiefd by drug screening methods with the phosphodiesterase **polypeptides** and polynucleotides as a target.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 38 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:133840 USPATFULL
TITLE: 13237, 18480, 2245 or 16228 novel human **protein kinase** molecules and uses

therefor

INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Rudolph-Owen, Laura A., Jamaica Plains, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Tsai, Fong Ying, Newton, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002068698	A1	20020606	
APPLICATION INFO.:	US 2001-910150	A1	20010718	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-219028P	20000718 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Carolyn A. Favorito, Morrison & Foerster LLP, Suite 500, 3811 Valley Centre Drive, San Diego, CA, 92130-2332
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 5427
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 13237, 18480, 2245 or 16228 nucleic acid molecules, which encode novel **protein kinase** family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 13237, 18480, 2245 or 16228 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 13237, 18480, 2245 or 16228 gene has been introduced or disrupted. The invention still further provides isolated 13237, 18480, 2245 or 16228 **proteins**, fusion **proteins**, antigenic **peptides** and anti-13237, -18480, -2245 or -16228 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 39 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:119590 USPATFULL
TITLE: 18431 and 32374, novel human **protein kinase** family members and uses therefor
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Silos-Santiago, Inmaculada, Cambridge, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002061573	A1	20020523	
APPLICATION INFO.:	US 2001-916790	A1	20010727	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-220644P	20000725 (60)

PRIORITY INFORMATION: US 2000-221543P 20000728 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Carolyn A. Favorito, Morrison & Foerster LLP, Suite 500, 3811 Valley Centre Drive, San Diego, CA, 92130-2332
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 4936
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 32374 or 18431 nucleic acid molecules, which encode novel **protein kinase** family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 32374 or 18431 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 32374 or 18431 gene has been introduced or disrupted. The invention still further provides isolated 32374 or 18431 **proteins**, fusion **proteins**, antigenic **peptides** and anti-32374 or -18431 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 40 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:78445 USPATFULL
TITLE: 2504, 15977, and 14760, novel **protein kinase** family members and uses therefor
INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, UNITED STATES
Meyers, Rachel A., Newton, MA, UNITED STATES
Curtis, Rory A.J., Southborough, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002042099	A1	20020411	
	US 6730491	B2	20040504	
APPLICATION INFO.:	US 2001-797039	A1	20010228	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-186061P	20000229 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, FISH & RICHARDSON P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 5985
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 2504, 15977, or 14760 nucleic acid molecules, which encode novel **protein kinase** family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 2504, 15977, or 14760 nucleic acid molecules,

host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 2504, 15977, or 14760 gene has been introduced or disrupted. The invention still further provides isolated 2504, 15977, or 14760 **proteins**, fusion **proteins**, antigenic **peptides** and anti-2504, 15977, or 14760 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 41 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:75569 USPATFULL
TITLE: 22012, human carboxypeptidase
INVENTOR(S): **Kapeller-Libermann, Rosana**, Chestnut Hill, MA, United States
MacBeth, Kyle J., Boston, MA, United States
Williamson, Mark, Saugus, MA, United States
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6369210	B1	20020409	
APPLICATION INFO.:	US 1999-345469		19990630	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Caputa, Anthony C.
ASSISTANT EXAMINER: Harris, Alana M.
LEGAL REPRESENTATIVE: Alston & Bird LLP
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 3275
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a newly identified human carboxypeptidase. The invention also relates to polynucleotides encoding the carboxypeptidase. The invention further relates to methods using the carboxypeptidase **polypeptides** and polynucleotides as a target for diagnosis and treatment in carboxypeptidase-related disorders. The invention further relates to drug-screening methods using the carboxypeptidase **polypeptides** and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the carboxypeptidase **polypeptides** and polynucleotides. The invention further relates to procedures for producing the carboxypeptidase **polypeptides** and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 42 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:60946 USPATFULL
TITLE: Novel human **protein kinases** and uses therefor
INVENTOR(S): **Meyers, Rachel**, Newton, MA, UNITED STATES
Kapeller-Libermann, Rosana, Chestnut Hill, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED

STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002034780	A1	20020321
	US 6638721	B2	20031028
APPLICATION INFO.:	US 2001-799875	A1	20010306 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-659287, filed on 12 Sep 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-182059P	20000211 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 58 Drawing Page(s)
LINE COUNT: 6018
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to novel kinase nucleic acid sequences and **proteins**. Also provided are vectors, host cells, and recombinant methods for making and using the novel molecules.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 43 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:54654 USPATFULL
TITLE: 46619, a novel human beta-ketoacyl synthase and uses thereof
INVENTOR(S): **Meyers, Rachel A.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002031815	A1	20020314
APPLICATION INFO.:	US 2001-892870	A1	20010626 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-214174P	20000626 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 4474
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated and novel nucleic acids molecules, designated beta-ketoacyl synthase nucleic acid molecules, which encode novel beta-ketoacyl synthase **polypeptides**. The

invention also provides antisense nucleic acid molecules, recombinant expression vectors containing beta-ketoacyl synthase nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a beta-ketoacyl synthase gene has been introduced or disrupted. The invention still further provides isolated beta-ketoacyl synthase **proteins**, fusion **proteins**, antigenic **peptides** and anti-beta-ketoacyl synthase antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 44 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2002:27601 USPATFULL

TITLE: 46743 and 27417, novel human acyltransferase family members and uses therefor

INVENTOR(S): **Meyers, Rachel A.**, Newton, MA, UNITED STATES
Macbeth, Kyle J., Boston, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES
Rudolph-Owen, Laura A., Jamaica Plain, MA, UNITED STATES
Tsai, Fong-Ying, Newton, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002016449	A1	20020207	
APPLICATION INFO.:	US 2001-817910	A1	20010326	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-192092P	20000324 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, FISH & RICHARDSON P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 5587
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids molecules, designated 46743 or 27417 nucleic acid molecules, which encode novel acyltransferase family members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 46743 or 27417 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 46743 or 27417 gene has been introduced or disrupted. The invention still further provides isolated 46743 or 27417 **proteins**, fusion **proteins**, antigenic **peptides** and anti-46743 or anti-27417 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 45 OF 47 USPATFULL on STN

ACCESSION NUMBER: 2002:16899 USPATFULL
TITLE: 50365, a novel hexokinase family member and uses therefor
INVENTOR(S): **Meyers, Rachel A.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002009779	A1	20020124	
APPLICATION INFO.:	US 2001-861801	A1	20010521	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-205508P	20000519	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, FISH & RICHARDSON P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 5137
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated 50365 nucleic acid molecules, which encode novel hexokinase members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 50365 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 50365 gene has been introduced or disrupted. The invention still further provides isolated 50365 **proteins**, fusion **proteins** , antigenic **peptides** and anti-50365 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 46 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:16897 USPATFULL
TITLE: 25552, a novel human methyltransferase family member and uses thereof
INVENTOR(S): **Meyers, Rachel A.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002009777	A1	20020124	
APPLICATION INFO.:	US 2001-816714	A1	20010323	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-191865P	20000324	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, FISH & RICHARDSON P.C., 225 Franklin Street, Boston, MA, 02110-2804

NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Page(s)
LINE COUNT: 5107
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated 25552 nucleic acid molecules, which encode novel ubiE methyltransferase members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 25552 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 25552 gene has been introduced or disrupted. The invention still further provides isolated 25552 **proteins**, fusion **proteins**, antigenic **peptides** and anti-25552 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L39 ANSWER 47 OF 47 USPATFULL on STN
ACCESSION NUMBER: 2002:12274 USPATFULL
TITLE: 25692, a novel human O-Methyltransferase family member and uses thereof
INVENTOR(S): **Meyers, Rachel A.**, Newton, MA, UNITED STATES
Williamson, Mark, Saugus, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002006653	A1	20020117	
APPLICATION INFO.:	US 2001-844468	A1	20010427	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-200632P	20000428 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LOUIS MYERS, FISH & RICHARDSON P.C., 225 Franklin Street, Boston, MA, 02110-2804
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 5200
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids molecules, designated 25692 nucleic acid molecules, which encode novel O-Methyltransferase members. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing 25692 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 25692 gene has been introduced or disrupted. The invention still further provides isolated 25692 **proteins**, fusion **proteins**, antigenic **peptides** and anti-25692 antibodies. Diagnostic methods utilizing compositions of the invention are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

FILE 'HOME' ENTERED AT 14:24:35 ON 14 MAR 2006

Searcher : Shears 571-272-2528

Searcher : Shears 571-272-2528

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     (FILE 'HOME' ENTERED AT 13:52:20 ON 14 MAR 2006)
               SET COST OFF

     FILE 'CAPLUS' ENTERED AT 13:52:34 ON 14 MAR 2006
L1             0 SEA ABB=ON  PLU=ON  PTA2204 OR PTA 2204

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     JICST-EPLUS, JAPIO' ENTERED AT 13:52:57 ON 14 MAR 2006
L2             0 SEA ABB=ON  PLU=ON  L1

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L3             2 SEA ABB=ON  PLU=ON  PTA2204 OR PTA 2204

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     FILE 'USPATFULL' ENTERED AT 13:54:00 ON 14 MAR 2006
               D L3 1-2 IBIB ABS

     FILE 'REGISTRY' ENTERED AT 13:54:00 ON 14 MAR 2006
               E SODIUM CHLORIDE/CN
L4           445 SEA ABB=ON  PLU=ON  SODIUM CHLORIDE ?/CN
               E SODIUM CITRATE/CN
L5             5 SEA ABB=ON  PLU=ON  ("SODIUM CITRATE"/CN OR "SODIUM
                 CITRATE (NA2O7C6H6)"/CN OR "SODIUM CITRATE (NA3C6D5O7)"/CN
                 OR "SODIUM CITRATE (NA3C6H5O7)"/CN OR "SODIUM CITRATE
                 (NAC6H7O7)"/CN)
L6           450 SEA ABB=ON  PLU=ON  L4 OR L5
               E PROTEIN KINASE/CN
L7          1698 SEA ABB=ON  PLU=ON  PROTEIN KINASE ?/CN

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L*** DEL 263761 S L4 OR (NA OR SODIUM)(W)(CL OR CHLORIDE) OR SALINE
L*** DEL  20942 S L5 OR (NA OR TRISODIUM OR SODIUM)(W)CITRATE OR CITRA
L*** DEL    916 S L8 AND (L9 OR L10)
L*** DEL     21 S L11 AND (HYBRIDIS? OR HYBRIDIZ?)
L*** DEL      1 S L12 AND SSC
L*** DEL      3 S L12 AND SDS]
L*** DEL      3 S L12 AND SDS
L*** DEL    908 S L8 AND L9
L*** DEL      5 S L15 AND L10
               D QUE L13
               D QUE L14
               D QUE L16
L*** DEL      7 S L13 OR L14 OR L16
               D 1-7 .BEVSTR

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     JICST-EPLUS, JAPIO' ENTERED AT 13:58:52 ON 14 MAR 2006
L*** DEL     93 S L12
L*** DEL     14 S L16

     FILE 'CAPLUS' ENTERED AT 13:59:46 ON 14 MAR 2006
               D QUE L12
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L*** DEL     908 S L8 AND L9
L*** DEL       5 S L13 AND L10
                 D QUE L12
                 D QUE L14
L*** DEL      24 S L12 OR L14
                 D 1-24 .BEVSTR
                 D QUE L9
L9         430653 SEA ABB=ON  PLU=ON  L4 OR (NA OR SODIUM)(W)(CL OR CHLORIDE)
                  OR NACL OR SALINE
L10         20942 SEA ABB=ON  PLU=ON  L5 OR (NA OR TRISODIUM OR SODIUM)(W)CIT
                 RATE OR CITRA
L11          1634 SEA ABB=ON  PLU=ON  L8 AND L9
L12             5 SEA ABB=ON  PLU=ON  L11 AND L10
L13          1642 SEA ABB=ON  PLU=ON  L8 AND (L9 OR L10)
L14            29 SEA ABB=ON  PLU=ON  L13 AND (HYBRIDIS? OR HYBRIDIZ?)
L15            28 SEA ABB=ON  PLU=ON  L14 AND (POLYPEPTIDE OR PEPTIDE OR
                 PROTEIN OR POLYPROTEIN)
                 D KWIC
                 D QUE
L*** DEL    1138 S L8(L)L9
L*** DEL       1 S L16(L)L10
L*** DEL      31 S L15 OR L12
L*** DEL       2 S L15 AND (CELSIUM OR DEGREE(1W)C)
                 D KWIC
L*** DEL       7 S L15 AND (SSC OR SDS)
L*** DEL       0 S SSC AND MEYERS ?/AU
                 D QUE L12
                 D QUE L14
L16            32 SEA ABB=ON  PLU=ON  L12 OR L14
                 D 1-32 .BEVSTR

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO' ENTERED AT 14:08:07 ON 14 MAR 2006
L17            16 SEA ABB=ON  PLU=ON  L12
L18          3927 SEA ABB=ON  PLU=ON  L8(L)(L9 OR L10)
L19            52 SEA ABB=ON  PLU=ON  L18(L)(HYBRIDIS? OR HYBRIDIZ?)
L20            52 SEA ABB=ON  PLU=ON  L19(L)(POLYPEPTIDE OR PEPTIDE OR
                 PROTEIN OR POLYPROTEIN)
L21            59 SEA ABB=ON  PLU=ON  L17 OR L20
L22            39 DUP REM L21 (20 DUPLICATES REMOVED)
                 D 1-39 IBIB ABS

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L23         16071 SEA ABB=ON  PLU=ON  L8(L)L9
L24          6106 SEA ABB=ON  PLU=ON  L23(L)L10
L25          5507 SEA ABB=ON  PLU=ON  L24(L)(HYBRIDIS? OR HYBRIDIZ?)
L26           527 SEA ABB=ON  PLU=ON  L8(S)L9
L27            23 SEA ABB=ON  PLU=ON  L26(S)L10
L28            21 SEA ABB=ON  PLU=ON  L27(S)(HYBRIDIS? OR HYBRIDIZ?)
L*** DEL     528 S L8(S)(L9 OR L10)
L*** DEL      41 S L29(S)(HYBRIDIS? OR HYBRIDIZ?)
L*** DEL      41 S (L28 OR L30)(S)(PEPTIDE OR POLYPEPTIDE OR PROTEIN OR POLY
L*** DEL      41 S L30 NOT L3
L*** DEL     145 S L21 AND MEYERS ?/AU
L*** DEL      27 S L30 AND WILLIAMSON ?/AU
L*** DEL      10 S L30 AND (KAPELLER? OR LIBREMANN?)/AU
L*** DEL      10 S L30 AND (KAPELLER? OR LIBERMANN?)/AU
L*** DEL       0 S L27 AND MEYERS ?/AU
L*** DEL     140 S L25 AND MEYERS ?/AU
```

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L*** DEL    5489 S L25 AND WASH?
L*** DEL    5465 S L25(L)WASH?
L*** DEL       0 S L30 AND MEYERS ?/AU
L29           21 SEA ABB=ON  PLU=ON  L28 NOT L3
                 D QUE L28
                 D L29 1-21 IBIB ABS

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO, USPATFULL' ENTERED AT 14:16:44 ON 14 MAR 2006
L30         2011 SEA ABB=ON  PLU=ON  "MEYERS R"?/AU
L31          498 SEA ABB=ON  PLU=ON  ("KAPELLER LIBERMANN R"? OR "LIBERMANN
                 KAPELLER R"? OR "LIBERMANN R"? OR "KAPELLER R"?)/AU
L32         3809 SEA ABB=ON  PLU=ON  "WILLIAMSON M"?/AU
L33           45 SEA ABB=ON  PLU=ON  L30 AND L31 AND L32
L34          151 SEA ABB=ON  PLU=ON  L30 AND (L31 OR L32)
L35           65 SEA ABB=ON  PLU=ON  L31 AND L32
L*** DEL     236 S (L33 OR L34 OR L35 OR L30 OR L31 OR L32) AND L14
L*** DEL     236 S L36 AND (PEPTIDE OR POLYPEPTIDE OR PROTEIN OR POLYPROTEI
L*** DEL     236 S L37 AND WASH?
L*** DEL     236 S L39 AND (ISOL OR ISOLAT?)
L*** DEL     236 S L39 AND PLASMID
                 D KWIC
L*** DEL     236 S L*** AND (CDNA OR (C OR COMPLEMENT?)(W)(DNA OR DEOXYRIBON
L36           47 SEA ABB=ON  PLU=ON  (L33 OR L34 OR L35) AND L14
L37           47 SEA ABB=ON  PLU=ON  L36 AND (PEPTIDE OR POLYPEPTIDE OR
                 PROTEIN OR POLYPROTEIN)
L38           47 DUP REM L37 (0 DUPLICATES REMOVED)
L39           47 SEA ABB=ON  PLU=ON  L38 AND WASH?
                 D 1-47 IBIB ABS

     FILE 'HOME' ENTERED AT 14:24:35 ON 14 MAR 2006


     FILE HOME

     FILE CAPLUS

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     FILE COVERS 1907 - 14 Mar 2006  VOL 144 ISS 12
     FILE LAST UPDATED: 13 Mar 2006  (20060313/ED)

     Effective October 17, 2005, revised CAS Information Use Policies apply
     They are available for your review at:

     http://www.cas.org/infopolicy.html

     FILE MEDLINE
      FILE LAST UPDATED: 11 MAR 2006 (20060311/UP).  FILE COVERS 1950 TO DA

      On December 11, 2005, the 2006 MeSH terms were loaded.
```

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
See also:

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.ht
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE BIOSIS
FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 9 March 2006 (20060309/ED)

FILE EMBASE
FILE COVERS 1974 TO 10 Mar 2006 (20060310/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

The updates on February 20 and 24, 2006, were incomplete due to a technical problem. The problem has been corrected, and the missing records were included in the update on March 3, 2006. If you received SDI results from the original updates on February 20 and 24, you will automatically be credited for the update that was rerun on March 3.

If you have any questions, please contact your STN Service Center.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE WPIDS
FILE LAST UPDATED: 10 MAR 2006 <20060310/UP>
MOST RECENT DERWENT UPDATE: 200617 <200617/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE, PLEASE VISIT:
http://www.stn-international.de/training_center/patents/stn_guide.pdf

>>> FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE
http://scientific.thomson.com/support/patents/coverage/latestupdates/

>>> FOR INFORMATION ON ALL DERWENT WORLD PATENTS INDEX USER GUIDES, PLEASE VISIT:
http://scientific.thomson.com/support/products/dwpi/

Searcher : Shears 571-272-2528

>>> FAST-ALERTING ACCESS TO NEWLY-PUBLISHED PATENT
DOCUMENTATION NOW AVAILABLE IN DERWENT WORLD PATENTS INDEX
FIRST VIEW - FILE WPIFV.
FOR FURTHER DETAILS:
http://scientific.thomson.com/support/products/dwpifv/

>>> THE CPI AND EPI MANUAL CODES WILL BE REVISED FROM UPDATE 200601.
PLEASE CHECK:
http://scientific.thomson.com/support/patents/dwpiref/reftools/classif

>>> PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ipc_reform.html
http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf <<<

FILE CONFSCI
FILE COVERS 1973 TO 25 May 2005 (20050525/ED)

CSA has suspended updates until further notice.

FILE SCISEARCH

FILE COVERS 1974 TO 9 Mar 2006 (20060309/ED)

SCISEARCH has been reloaded, see HELP RLOAD for details.

FILE JICST-EPLUS
FILE COVERS 1985 TO 13 MAR 2006 (20060313/ED)

THE JICST-EPLUS FILE HAS BEEN RELOADED TO REFLECT THE 1999 CONTROLLED TERM (/CT) THESAURUS RELOAD.

FILE JAPIO
FILE COVERS APR 1973 TO OCTOBER 27, 2005

>>> GRAPHIC IMAGES AVAILABLE <<<

>>> NEW IPC8 DATA AND FUNCTIONALITY NOT YET AVAILABLE IN THIS FILE.
USE IPC7 FORMAT FOR SEARCHING THE IPC. WATCH THIS SPACE FOR FURTHE
DEVELOPMENTS AND SEE OUR NEWS SECTION FOR FURTHER INFORMATION
ABOUT THE IPC REFORM <<<

FILE USPATFULL
FILE COVERS 1971 TO PATENT PUBLICATION DATE: 14 Mar 2006 (20060314/PD)
FILE LAST UPDATED: 14 Mar 2006 (20060314/ED)
HIGHEST GRANTED PATENT NUMBER: US7013485
HIGHEST APPLICATION PUBLICATION NUMBER: US2006053519
CA INDEXING IS CURRENT THROUGH 14 Mar 2006 (20060314/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 14 Mar 2006 (20060314/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Dec 2005
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Dec 2005

FILE REGISTRY
Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 13 MAR 2006 HIGHEST RN 876655-59-3
DICTIONARY FILE UPDATES: 13 MAR 2006 HIGHEST RN 876655-59-3

Searcher : Shears 571-272-2528

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

****
*
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added, *
* effective March 20, 2005. A new display format, IDERL, is now *
* available and contains the CA role and document type information. *
*
****

Structure search iteration limits have been increased. See HELP SLIMI for details.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

Searcher : Shears 571-272-2528

```
=> d his; d cost

     (FILE 'HOME' ENTERED AT 13:52:20 ON 14 MAR 2006)
               SET COST OFF

     FILE 'CAPLUS' ENTERED AT 13:52:34 ON 14 MAR 2006
L1             0 S PTA2204 OR PTA 2204

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO' ENTERED AT 13:52:57 ON 14 MAR 2006
L2             0 S L1

     FILE 'USPATFULL' ENTERED AT 13:53:10 ON 14 MAR 2006
L3             2 S L1

     FILE 'CAPLUS' ENTERED AT 13:53:59 ON 14 MAR 2006

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO' ENTERED AT 13:53:59 ON 14 MAR 2006

     FILE 'USPATFULL' ENTERED AT 13:54:00 ON 14 MAR 2006

     FILE 'REGISTRY' ENTERED AT 13:54:00 ON 14 MAR 2006
               E SODIUM CHLORIDE/CN
L4           445 S SODIUM CHLORIDE ?/CN
               E SODIUM CITRATE/CN
L5             5 S E3-7
L6           450 S L4 OR L5
               E PROTEIN KINASE/CN
L7          1698 S PROTEIN KINASE ?/CN

     FILE 'CAPLUS' ENTERED AT 13:55:14 ON 14 MAR 2006
L8        165813 S L7 OR PROTEIN KINASE

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO' ENTERED AT 13:58:52 ON 14 MAR 2006

     FILE 'CAPLUS' ENTERED AT 13:59:46 ON 14 MAR 2006
L9        430653 S L4 OR (NA OR SODIUM)(W)(CL OR CHLORIDE) OR NACL OR SALIN
L10        20942 S L5 OR (NA OR TRISODIUM OR SODIUM)(W)CITRATE OR CITRA
L11         1634 S L8 AND L9
L12            5 S L11 AND L10
L13         1642 S L8 AND (L9 OR L10)
L14           29 S L13 AND (HYBRIDIS? OR HYBRIDIZ?)
L15           28 S L14 AND (POLYPEPTIDE OR PEPTIDE OR PROTEIN OR POLYPROTEIN
L16           32 S L12 OR L14

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO' ENTERED AT 14:08:07 ON 14 MAR 2006
L17           16 S L12
L18         3927 S L8(L)(L9 OR L10)
L19           52 S L18(L)(HYBRIDIS? OR HYBRIDIZ?)
L20           52 S L19(L)(POLYPEPTIDE OR PEPTIDE OR PROTEIN OR POLYPROTEIN)
L21           59 S L17 OR L20
L22           39 DUP REM L21 (20 DUPLICATES REMOVED)

     FILE 'USPATFULL' ENTERED AT 14:11:01 ON 14 MAR 2006
L23        16071 S L8(L)L9
```

Searcher : Shears 571-272-2528